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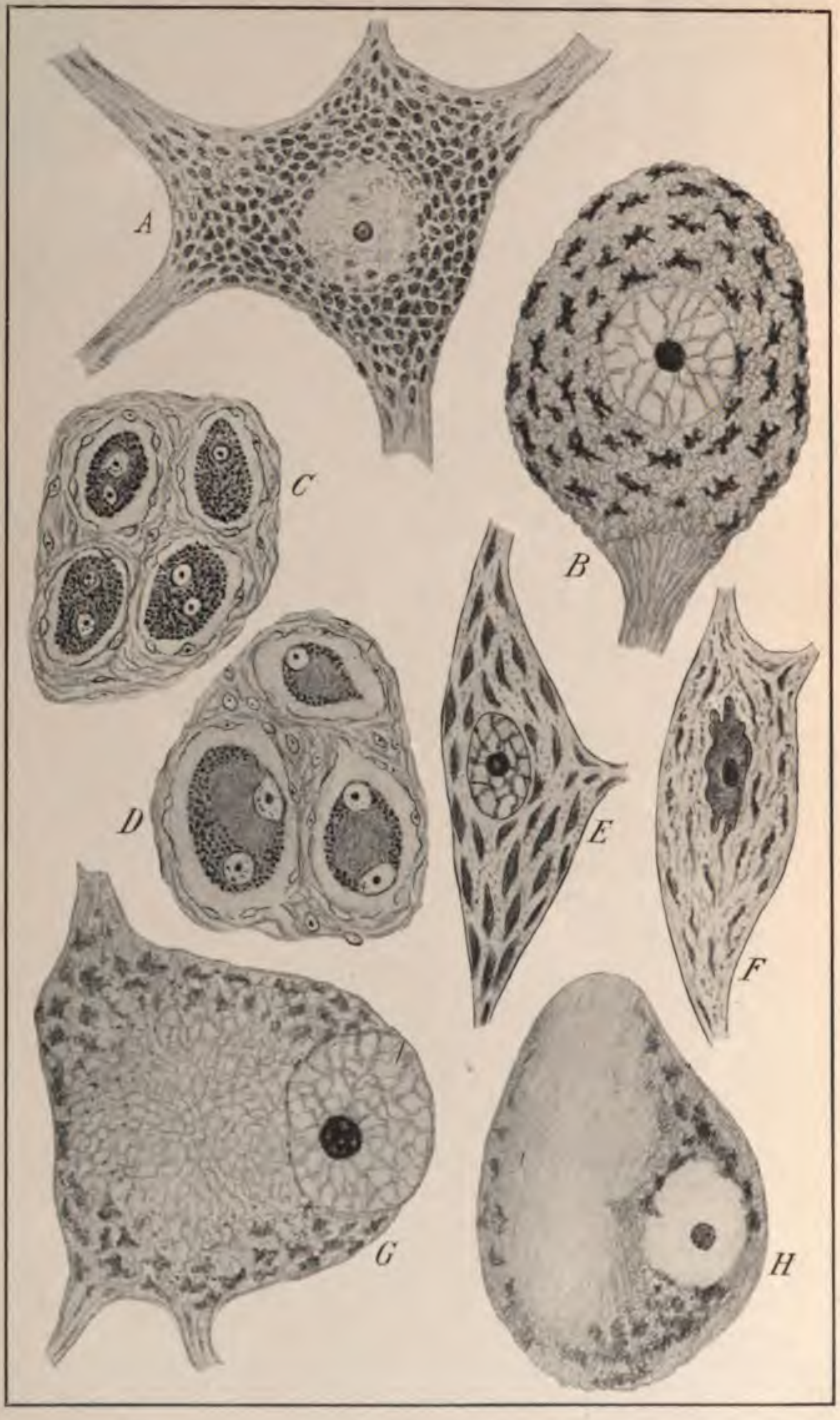
PLATE I.



A. Normal central neurone; Nissl stain (Flatau). *B.* Normal peripheral neurone (Ewing). *C.* Normal neurones at rest. *D.* Neurones exhausted by work (Vas). *E.* Normal neurone. *F. Exhausted neurone (Hodge).* *G H.* Degenerated neurones (Ewing).

ORGANIC
NERVOUS DISEASES.

BY

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"ATLAS OF NERVE CELLS."

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26 PLATES IN COLORS AND MONOCHROME.

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PREFACE.

THE investigations and discoveries in neurology during the past ten years have made the diagnosis of nervous diseases less difficult and more exact, so that it is now possible to trace any symptom presented by a patient to some disturbance of function in a definite nerve centre or nerve tract. And as the knowledge of these centres and tracts has grown, as their location and action have been made clear, the meaning of symptoms previously perplexing has become plain.

Accuracy of diagnosis has led in turn to precision in treatment, and especially to the application of surgical measures for the relief of diseases formerly considered incurable. There are few departments of medicine in which more remarkable progress has been made than in neurology; and there is none that lies so openly on the borderland between medicine and surgery. The surgical successes in the treatment of nervous affections are among the most brilliant of recent achievements in this domain.

The mass of pathological and clinical material that has accumulated in my possession during the past twenty years in the practice of this specialty has seemed worthy of analytical study, and has led me to many definite conclusions. It is upon this material to a large degree that the statements presented in this book are based. While the extensive literature of neurology has been carefully sifted, its facts collated, and its theories considered, the endeavor has been made to utilize personal observation and experience in the presentation of each subject.

It has been my object to make clear both to students and to practitioners the principles upon which the location and diagnosis of

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nervous diseases rest, and to put before them in a definite manner the appropriate medical and surgical treatment and its results.

For the cordial co-operation of Dr. F. R. Bailey in the preparation of pathological material; of Dr. Edward R. Leaming and Dr. R. H. Cunningham in photographic work, and of Dr. Smith Ely Jelliffe in reading the proof, I desire to express sincere thanks.

M. ALLEN STARR.

5 WEST FIFTY-FOURTH STREET, NEW YORK, 1903.

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ORGANIC NERVOUS DISEASES.

CHAPTER I.

THE STRUCTURE OF THE NERVOUS SYSTEM.

I. The Cerebro-spinal Nervous System. Neurones, Central and Peripheral. Dendrites. Axones. Terminal Tassels. Varieties of Neurones. Nutrition and Pathology of Neurones. II. The Sympathetic Nervous System.

THE nervous system is divided into two parts, the cerebro-spinal system and the sympathetic system.

I. THE CEREBRO-SPINAL NERVOUS SYSTEM.

The cerebro-spinal nervous system consists of two large central organs, the brain and spinal cord, and of a very extensive and widespread peripheral system of nerves which bring these central organs into communication with every part of the body. Inasmuch as every form of functional activity in the organism requires direction in accordance with the needs of each organ and also of the entire body, it is evident that a central regulating station in perfect connection with all the different parts is a necessity. The cerebro-spinal nervous system supplies this need. Its function consists in the reception of impressions and the response to these impressions by actions that are performed in a properly ordered manner adapted to an end. Incidentally impressions and actions are registered, so that recollection is possible and repetition is easy. These acts may be unconscious and automatic, or conscious and voluntary, according to the parts of the nervous system which are called into play by the impression. But, whether conscious or unconscious, the action is always one of control of the lower mechanisms of the body; of the sensory organs, and of the voluntary and involuntary muscles; of the circulatory and respiratory systems; of the secretory glands, and of the digestive and reproductive systems. Thus the nervous system may be considered as the governing and controlling system of the entire organism.

The central organs of the nervous system are made up of gray and white matter, whose visible difference led older anatomists to attach much importance to their varying arrangement. The gray matter was known to be made up of cells, and the white matter of nerve fibres transmitting impulses to and from the cells. But recent investigation has demonstrated that gray and white matter are merely different parts of elementary bodies, termed neurones, which make up the nervous system, and that the study of these neurones, of their structure, and of their connections, whether they lie singly or in groups, as in the spinal cord, or in masses, as in the basal ganglia of the brain, or spread out in layers, as in the cerebral cortex, is a matter of the greatest importance from every point of view. It is found that the white matter and the nerves are merely the distal portions of these neurones, and are not to be studied apart from the central part or body which forms the gray matter. It is therefore the chief object of the anatomist to-day to establish the situation of the neurone body and the course and termination of the neurone branches, their mutual relations, and their manner of action and interaction.

The neurones are held together by neuroglia and connective tissue, and are amply supplied with bloodvessels and lymphatics.

Classes of Neurones. There are two classes of neurones different in their origin in embryonal life. The first class develops in the central nervous system. The second class develops in the ganglia, which lie upon the nerves.

I. The First or Central Class of Neurone. The neurone consists of a cell body and its projecting branches. These are of two kinds, dendrites and axones.

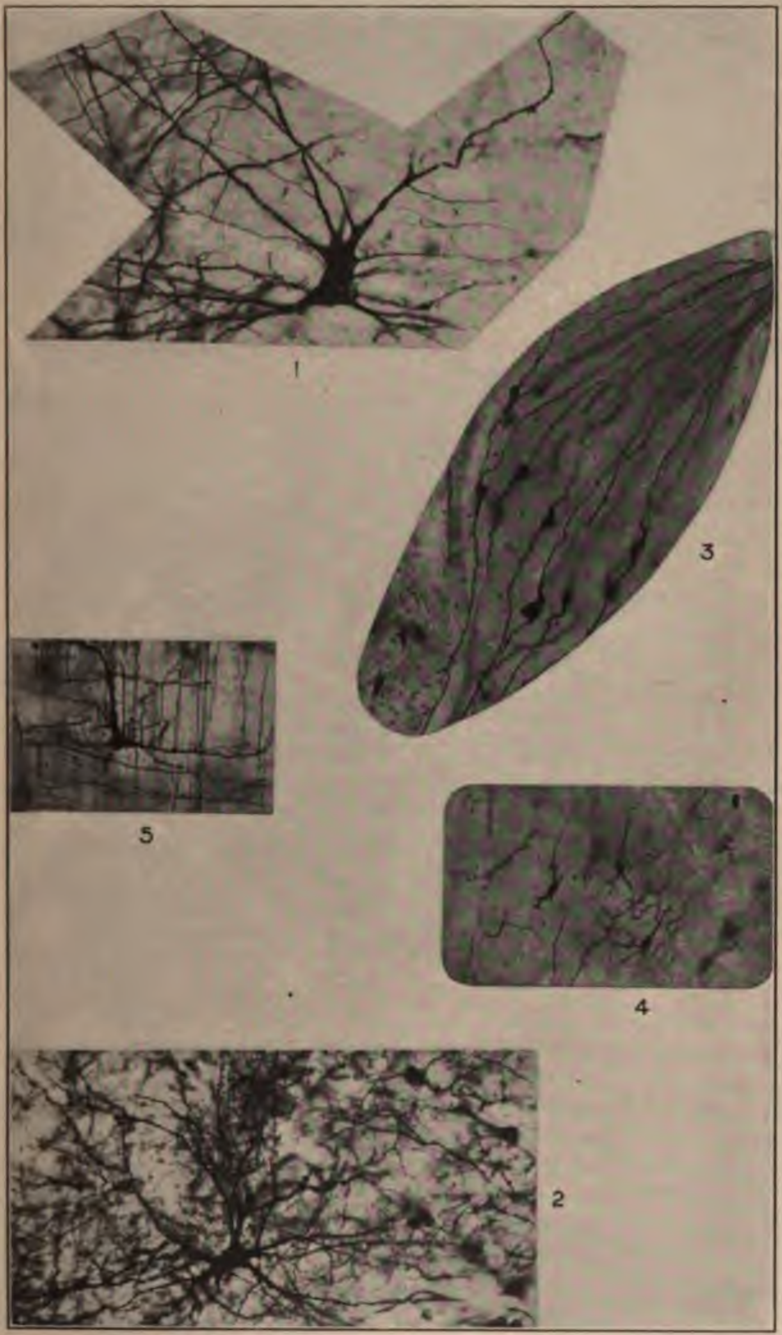
The cell body has various shapes in different parts of the nervous system, being spherical, pyramidal, and polygonal, as shown in Fig. 1. The actual structure of the body is shown in Plate I., *A*, which demonstrates a protoplasmic mass unstained, a nucleus and nucleolus and a large number of granular bodies which are more or less regularly arranged in the protoplasm about the nucleus. These bodies are stained by basic aniline dyes and have been termed Nissl bodies, chromophile bodies, stainable substance, or tigroid. Nissl bodies are made up of phospho-albumin, and it is supposed that this exists in solution in the living cell among the other ingredients of the protoplasm, but is precipitated by the fixative and then stained by the dye. The cell body varies greatly in size in different parts of the nervous system; it is from 4 to 135 microns in diameter. Through the cell body fine fibrils have been shown to pass in many directions from and to the branching processes[1] (Fig. 2), but some consider that the cell body is made up of a reticulum, and that these fibrils are artefacts.

The dendrites are branching protoplasmic processes of the cell body and resemble it in structure. They are very numerous in some

---

[1] The structure of the neurone body is still a matter of discussion. The reader is referred to Barker's Nervous System, Appleton & Co, 1898; to Ewing, Studies on Ganglion Cells, Archives of Neurology and Psychopathology, 1898, vol. i. p. 263; to Bailey, Morphology of Ganglion Cells, Journal of Experimental Medicine, 1901, vol. v. p. 550.

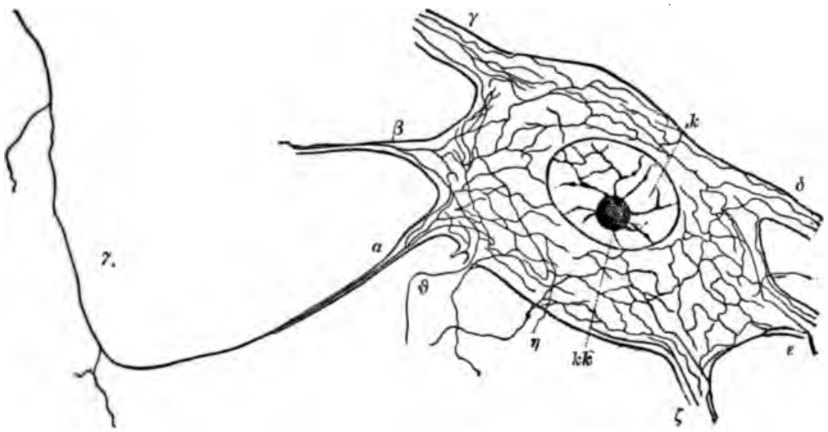
FIG. I.



Neurones. 1. Anterior horn of spinal cord. 2. Optic thalamus. 3. Posterior spinal ganglion (chick embryo). 4. Cerebellum. 5. Cerebral cortex, surface layer.

neurones. Near the cell body they are thick, but as they extend away they divide and subdivide, resembling the roots of a tree, until they end in fine fibrils. The dendrites vary in length, some neurones having one very long dendrite and many short ones. These variations are shown in Fig. 1. They pursue a tortuous course and are very irregular in their size, shape, and branching. The surface of the dendrite is rough and often nodular, and appears to be covered with small granules called buds or gemmules. Within the dendrite fine filaments lie which pass into the cell body, and through it into other dendrites or into the axone. It is supposed that the dendrites collect and transmit nervous impulses to the cell body; and as they are numerous and long it is evident that impulses from many sources may reach any one cell body.

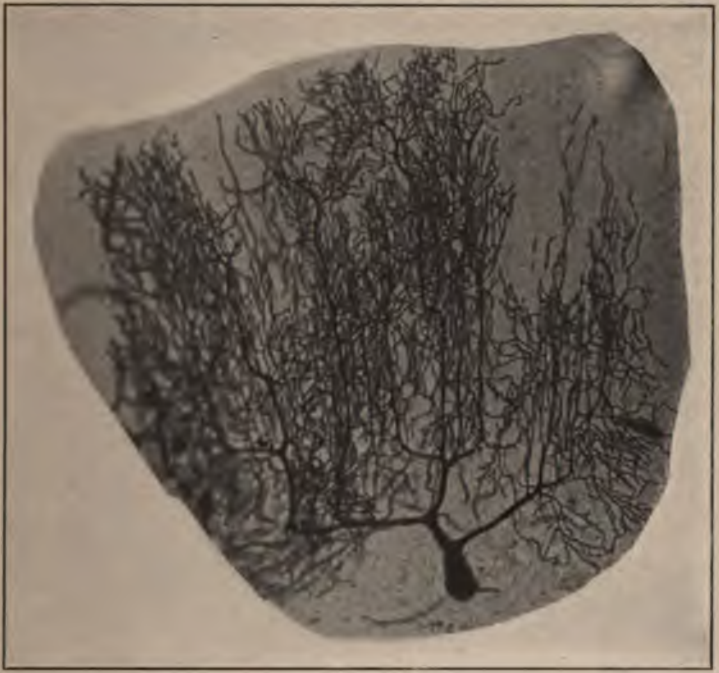
FIG. 2.



Large pluripolar ganglion cell of the ventral paramedian field of the abdominal cord of *Lumbricus*. A primitive fibril is seen on its way to a ganglion cell becoming thinner, owing to the emission of the side fibrils. A very complicated intracellular reticulum of neuro-fibrils is to be made out in the formation of which the primitive fibrils of all the processes take part. *kk*, nucleolus; *k*, nucleus. α and β are processes containing one primitive fibril in each, which arriving in the cell body split up into several bifurcating neuro-fibrils. The primitive fibril in α can be followed for a very long distance, being thicker at a distance from the cell. The coarse longitudinal processes γ and δ contain a large number of primitive fibrils which, as far as they can be followed, do not unite to one fibril. (After S. Apathy, Mitth. aus der zool. St. zu Neapel, 1897, Bd. xii., Ht. 4, Taf. xxvii., Fig. 7.)

The axone or axis-cylinder process of the neurone is a single, long branch, arising from a cone-like projection of the cell body. This cone-like projection, as well as its base within the cell body, contains no chromophile substance, but fine fibrils can be found within it by acid stains. It is supposed to transmit nervous impulses from the cell. It is straight and smooth in contour, and uniform in size. Though in its course within the central organs it gives off very fine collateral filaments at right angles to its direction, it does not branch in the nerve until it reaches its very end, some distance from the cell body. There it divides into a number of little end fibrils resembling a tuft or tassel. (Fig. 5.) In the central organs it is possible that these fibrils enter the dendrites of other neurones. It is possible that they end in free terminations which merely touch these dendrites. The collaterals terminate in the same manner. The termination of the axone in muscles is in the so-called muscle plate. In the other organs it ends in a fine plexus of nerve filaments. The axone is made up of many fine fibrils packed together, which are gathered from the cell body and from its dendrites. Fig. 6 shows this structure. The size of the axone at its exit from the cell body varies in different neurones. Some axones extend long distances, as from the cortex of the brain to the lowest part of the spinal cord. This variety is known as Type I. of Golgi. (Fig. 7.)

FIG. 3.

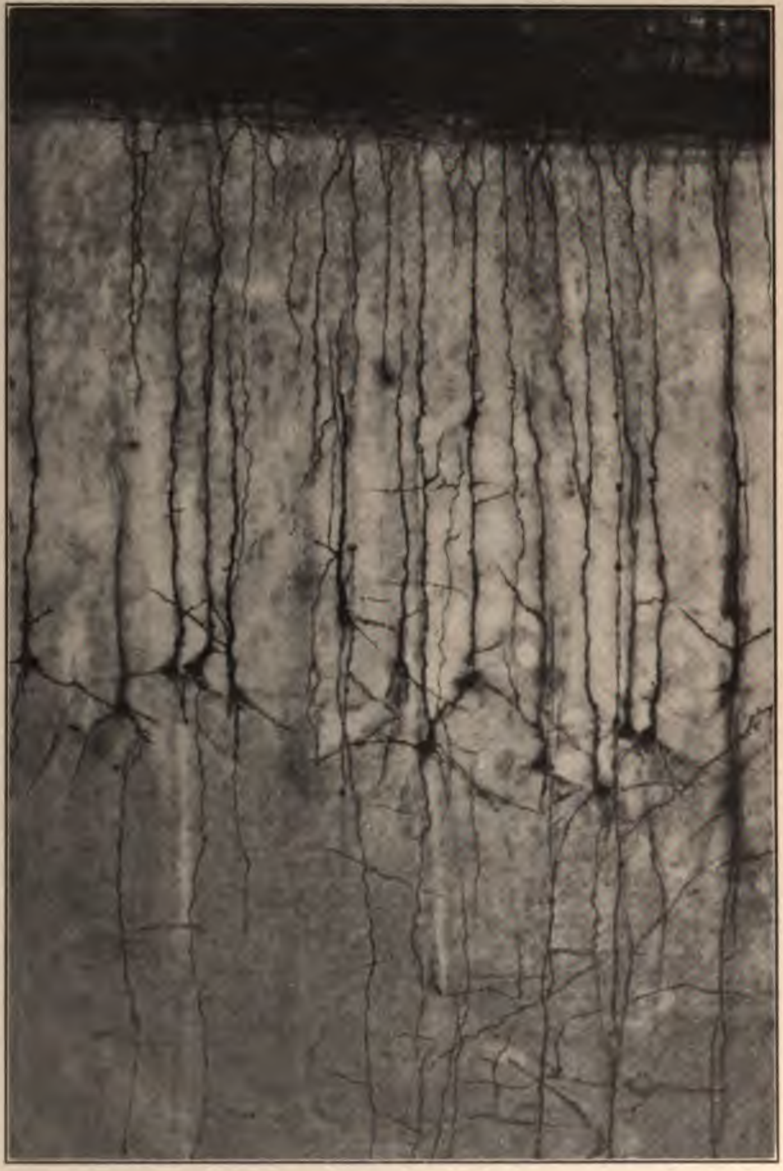


Purkinje neurone of cerebellum. Many dendrites. One axone projecting from the body.

Some axones terminate near the cell body, dividing and subdividing into a sort of network. This variety is known as Type II. of Golgi. (Fig. 8.) Between these extremes all varieties are to be found.

When the long axones leave the gray matter they are insulated from each other by receiving a medullary sheath or fatty coating held in place by a thin membrane, but this they lose at their termination. In Fig. 7 this is shown. Some axones on nearing their termination appear to split in two in a Y-shaped manner and each division has its own end tassel. If the nervous impulse passes out from the cell

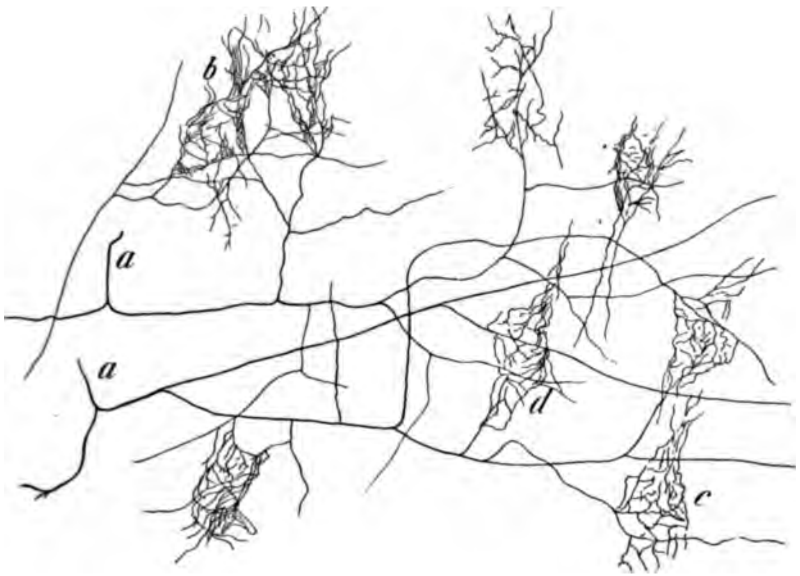
Fig. 4.



Neurones of the cerebral cortex, second layer. The long dendrite extends to the surface layer. Smaller dendrites go out from the body. The axone passes downward from the body, giving off collaterals.

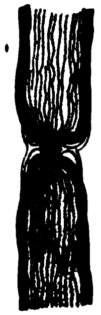
body along the axone it is evident that it may reach many different destinations, because of the different places of ending of the collaterals and of the terminal filaments. The terminal filaments of the axones are never continuous with other axones; but impulses are probably conveyed from one neurone to another by the contact of axones with adjacent dendrites or with other cell bodies.

FIG. 5.



The terminal fibres of the axones. *a*. Axone bifurcating near its termination. *b*, *c*, *d*. Terminal brushes. (Ramon y Cajal, Studien über die Hirnrinde des Menschen, I., 55.)

FIG. 6.

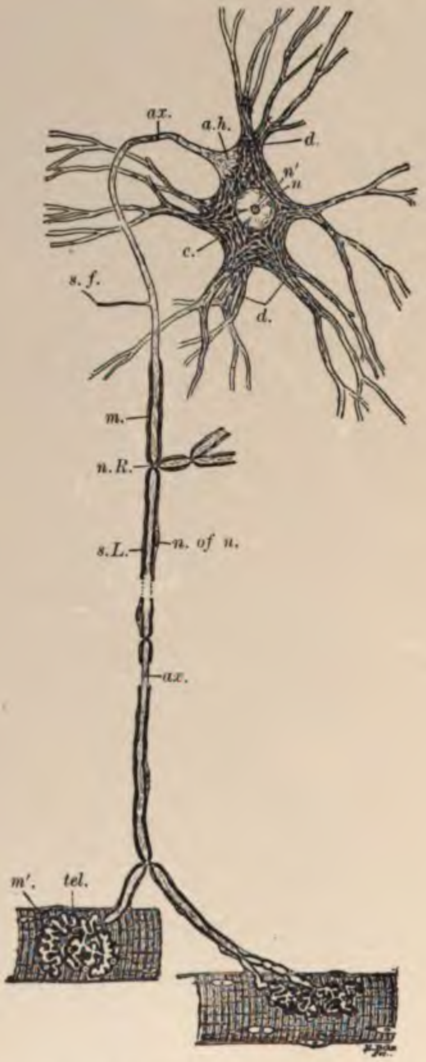


Axone from the sciatic nerve of a frog, showing fibrillary structure and a node of Ranvier. (Verworn.)

The nutrition of both axones and dendrites is controlled by the cell body, for they atrophy when separated from it. Nerves are the axones of neurones collected into bundles and held together by connective tissue.

II. The Second or Peripheral Class of Neurone. This class belongs to the sensory part of the nervous system. It develops originally in the posterior spinal ganglia that lie outside of the spinal cord and in the homologous ganglia that are in connection with the sensory cranial nerves and also in the ganglia of the sympathetic system. The sensory neurone in an early stage of development is bipolar, a branch coming out of each end of the cell body. (Fig. 1, *3*.) Later the two branches appear to be fused together for a short distance, and thus in the adult the sensory neurone is pear-shaped and appears to send out a single axone which divides into two branches, passing in opposite directions. One of these branches finds its way outward to the periphery of the body, forming a sensory nerve, and

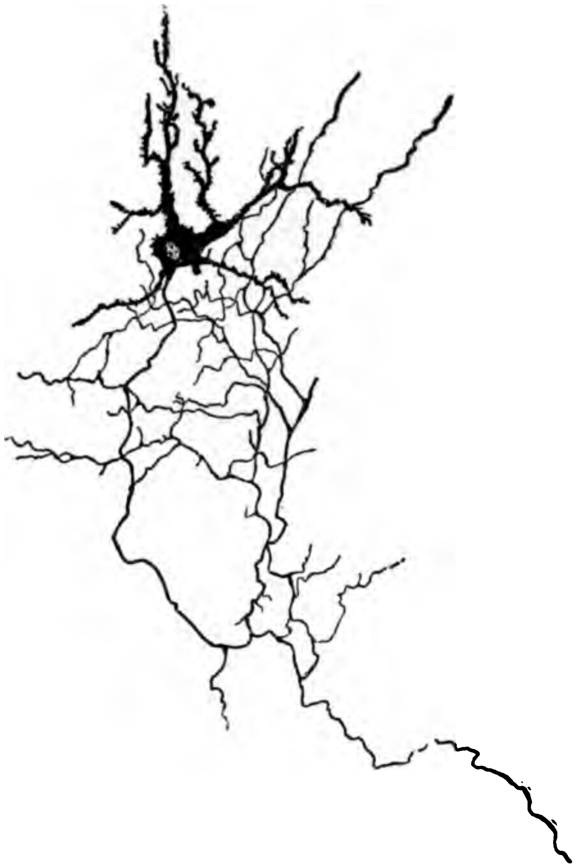
FIG. 7.



Scheme of central motor neurone. (I. type of Golgi.) The motor cell body, together with all its protoplasmic processes, its axis-cylinder process, side fibrils, or collaterals, and end ramifications, represent parts of a single cell or *neurone*. *a.h.*, axone-hillock devoid of Nissl bodies, and showing fibrillation; *ax.*, axis cylinder or axone. This process, near the cell body, becomes surrounded by myelin, *m.*, and a cellular sheath, the neurilemma, the latter not being an integral part of the neurone; *c.*, cytoplasm showing Nissl bodies and lighter ground substance; *d.*, protoplasmic processes (dendrites) containing Nissl bodies; *n.*, nucleus; *n'.*, nucleolus; *n.R.*, node of Ranvier; *s.f.*, side fibril; *n. of n.*, nucleus of neurilemma sheath; *tel.*, motor end plate or telodendron; *m'.*, striped muscle fibre; *s.L.*, segmentation of Lantermann. (Barker.)

terminates in a fine brush-like expansion of filaments in the skin or in the tactile corpuscles. The other branch finds its way inward through the posterior nerve root into the spinal cord or brain axis, where it bifurcates, one portion passing downward and the other portion upward in the posterior columns of the spinal cord, or in the formatio reticularis of the brain axis. As these main branches pass up and down within the cord they give off at right angles little collaterals, and these collaterals, together with the main branches, terminate in brush-like extremities within the gray matter of the posterior horn, either near their entrance or as far from their entrance as the posterior nuclei of the medulla oblongata, or at various levels in the brain axis. The sensory axone never terminates in a cell in the spinal cord or brain axis. It ends in brush-like terminations or tassels.

FIG. 8.



Golgi's cell of Type II. from the dorsal horn of the gray matter of the spinal cord of the newborn mouse. Even in such a neurone the axone is very easily distinguishable from the dendrites. The latter are only represented in part in the illustration. (After von Lenhossék.)

In this form of neurone the cell body is situated about one-half way between the terminal extremities of its two great branches, and this fact of the interposition of the body in the course of a nerve tract, which, from the nature of the case, must be a continuous tract, suggests that the function of the cell body is a trophic one. We have proof also that an axone is not always centrifugal in the direction of its impulses.

The structure of the cell body in this class of neurones is not different from that in the first class; but the chromophile granules are arranged about the nucleus in a series of concentric rings. (See Plate I., *B.*)

Varieties of Neurones. Neurones have been classified into:

1. Primary neurones, whose cell bodies lie in the central nervous system or ganglia and whose axones extend thence to some peripheral part of the organism, to a muscle, or to a gland, or to the skin, or to some sensory organ, and
2. Secondary neurones, whose cell bodies lie in the central nervous system and whose axones extend to some other part of the central nervous system, to terminate about a primary neurone or about another secondary neurone.

Neurones have also been classified according to their function into

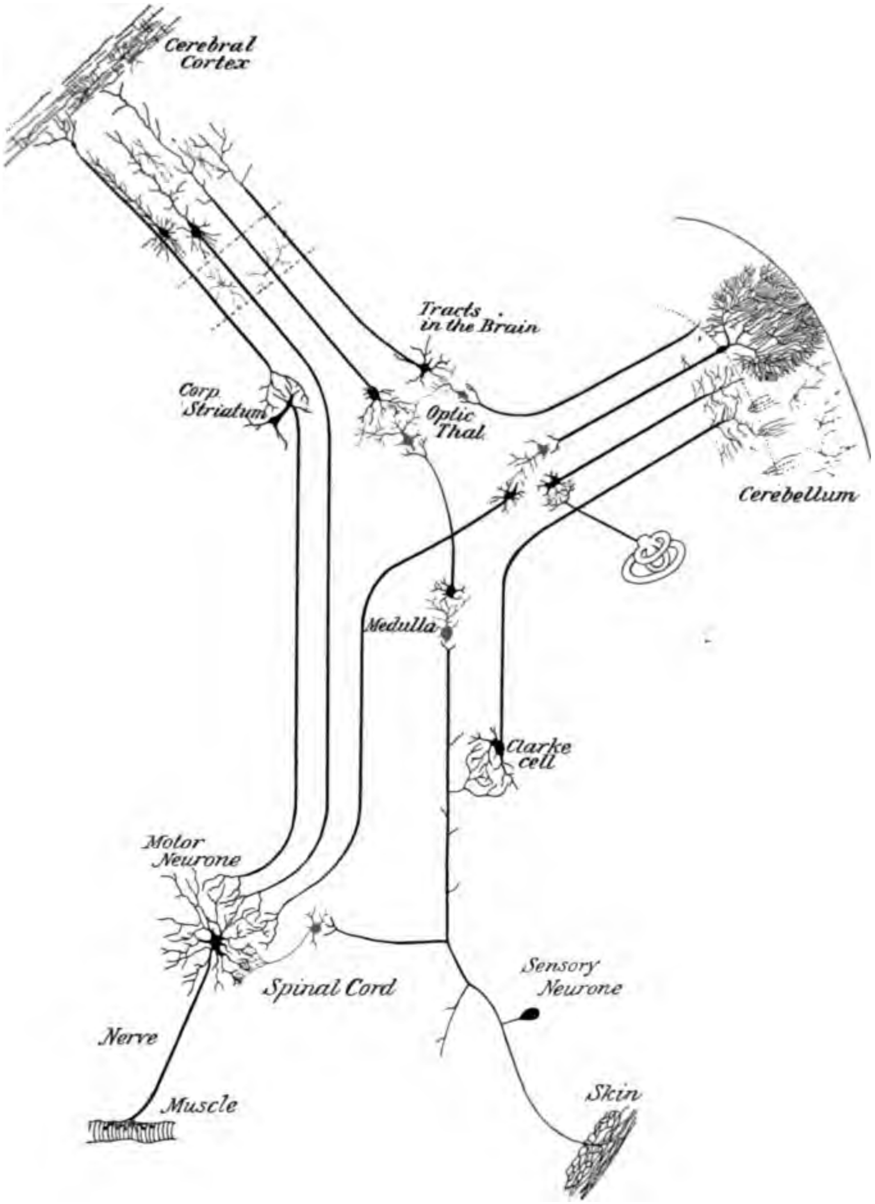
1. Centrifugal: (*a*) motor, (*b*) secretory, (*c*) trophic.
2. Centripetal or sensory.
3. Intrinsic or association.

The diagram (Plate II.) illustrates these various neurones and their relations to one another.

Reflex acts may be performed by the primary neurones, but all conscious sensations or voluntary acts involve the action of both primary and secondary neurones acting together. This action may be illustrated by the analysis of a simple sensation, like the impact of a particle of dust in the eye, which causes a wink, a flow of tears, a conscious pain, and a voluntary effort to remove the particle. The sensation comes in from the eye along the primary sensory neurone, is transmitted (*a*) to the brain axis, where it reaches the primary motor neurone, setting up the centrifugal reflex acts of a wink and of secretion of tears, and (*b*) to the secondary sensory neurone, which transmits it upward to the brain. This secondary neurone may terminate about another secondary or association neurone, which then sends the impulse on to a secondary motor neurone, and this in turn sends its impulse down to the primary neurone, which transmits it to the muscle, causing the removal by the hand of the particle.

There are many more complex acts, either automatic, like the act of breathing or sneezing; or voluntary, like the act of walking or talking, which call into play a vast number of neurones—centripetal, intrinsic, centrifugal—which cannot be so easily analyzed. But however complex the act, the physical basis of all nervous and mental activity is the interaction of a series of neurones.

PLATE II.



The Relations of the Neurones.

The association neurones are shown in red. A sensory impression from the skin reaches the spinal cord, where it may excite the motor neurone; or the Clarke cell, and through it the cerebellum; or the medulla, optic thalamus and cerebral cortex, causing automatic responses and conscious perceptions. A sensory impression from the semicircular canals may reach the cerebellum, causing automatic acts of equilibrium. Cerebellar impulses may be sent to the motor neurone by way of the neurones of Deiters in the medulla, producing acts of balancing, or to the cerebrum by way of the optic thalamus, causing perceptions of position. Cerebral impulses may be sent directly to the motor neurone or indirectly through the corpus striatum, producing voluntary or automatic acts. The motor neurone sends its nerve to the muscle.

The arrangement of these neurones and their mutual relations will be considered in connection with the studies of diseases of the various parts of the nervous system. In that connection also the vascular supply of the nervous system will be described.

The Nutrition of the Neurone. In order that these neurones may do their work they must have a normal structure and a normal capacity for sustaining their nutrition when in activity. When a neurone is made to work it undergoes certain manifest changes. Hodge[1] was the first to describe alterations in the form and appearance of the cell and of its nucleus consequent upon its activity. There is a general diminution in the size of the cell, a lessened power to absorb staining substances, which may be taken as a proof of the using up of its own substance and also changes in the appearance of the nucleus, which is decreased in size, and changes from a smooth and rounded outline to a jagged and irregular one. As the cell becomes changed in its structure by constant work, it becomes more and more exhausted, so that finally there comes a time when it is no longer capable of sending out impulses, and requires a period of rest to make up what it has lost of form and to regain a store of energy. Vas[2] and Mann[3] have described these physiological changes more exactly. Vas showed that mild stimulation of a cell caused a swelling of its body and of the nuclei and a clearing up of the central part of the cell by an apparent movement of the chromatin bodies to its periphery. Mann showed that functional activity of the cell is accompanied by an increase in the size due to imbibition of the lymph lying in the cavity about the cell, so that the cell at work fills up the cavity in which it lies. When activity goes on to the point of fatigue, then a shrivelling of the cell begins, first in the nucleus, then in the body. At the same time changes go on in the chromatin. During the period of activity the chromatin material is used up, so that a fatigued cell does not absorb staining material as does a cell at rest. In an exhausted cell the only stain is in and about the nucleus. In Plate I. these changes are shown. These results have been reached by stimulating cells to work in living animals either by electricity or by keeping up movements, such as running, or by exposing one eye to the light while the other was kept dark, and then contrasting the appearance of the cells made to work with those that were kept at rest. When a stimulated cell is allowed to rest it gradually resumes its original appearance; but the period of rest must be adequate. During the period of reconstruction the chemical activities going on in the cell are numerous, and its power of assimilation of material furnished to it by the blood must be increased. If, however, anything interferes with this increased nutrition, such as an imperfect supply of blood or a deleterious substance of any kind in the blood, then the process of building up fails to take place, and the neurone cannot resume its function.

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[1] **Hodge.** American Journal of Psychology, vol. ii. p. 39.

[2] **Vas.** Ueber den Bau des Chromatins in den lymphatischen Ganglien, Archiv. für Mikroscopische Anatomie, 1892, Heft 3, p. 375.

[3] **Gustav Mann.** Journal of Anatomy and Physiology, October, 1894.

The Pathology of the Neurone. The fundamental fact at the basis of all forms of nervous disease is some change in the power of the neurone to continue its normal functions. This may be of a temporary character, whose nature is as yet not understood, and not attended by visible alterations of form; and may be succeeded by a resumption of natural action. This is the hypothetical basis of functional nervous diseases. But the loss of power in the neurone may also be due to damage more or less grave to its structure, visible through the microscope, and often incapable of repair. This is the basis of organic diseases of the nervous system. The varieties will be carefully considered in connection with the various diseases studied. It is always to be remembered that the pathological changes which go on in the neurone are in no sense distinct from those occurring in the cells of other organs; but the exact appearances are determined by the structure of the neurone as different from that of other cells. We are at present able to see changes in the size and shape of the cell body and of its nucleus and nucleolus. We can distinguish, by Nissl's method of staining, changes in the chromophile bodies within the cell which consist in a gradually progressing disintegration of these bodies until they are resolved into a fine dust, and then entirely disappear, leaving the cell body a homogeneous, unstained mass. The protoplasmic substance in which these chromophile bodies lie also undergoes degenerative changes in disease; but our staining methods are not adequate to show this. We can see a displacement of the nucleus of the cell body to one side as the chromophile bodies disappear, which indicates either a solution of the substance about the nucleus, and its consequent sinking by gravitation from lack of support, or an active migration. We can see the development of vacuoles within the cell body, indicating an absorption of both protoplasmic and chromophile débris. And we can see a gradual shrivelling of the cell body until its place is taken by a granular scar. The dendrites and the axone share in the pathological processes within the cell body, showing similar evidences of disintegration and destruction, or the axone alone may undergo these changes in lighter grades of atrophy. All forms of neurones appear to undergo similar pathological alterations. These may be classed together, no matter what their cause, as parenchymatous inflammatory processes or as parenchymatous degeneration. There are certain chemical changes which attend degeneration in the neurones which have recently been determined by Mott.[1] He has ascertained that when nervous tissue degenerates, protagon or lecithin breaks up into choline, glycero-phosphoric acid, and stearic acid. The protagon is converted into a fat of a different composition, the phosphoretted fat being changed into a non-phosphoretted neutral fat.

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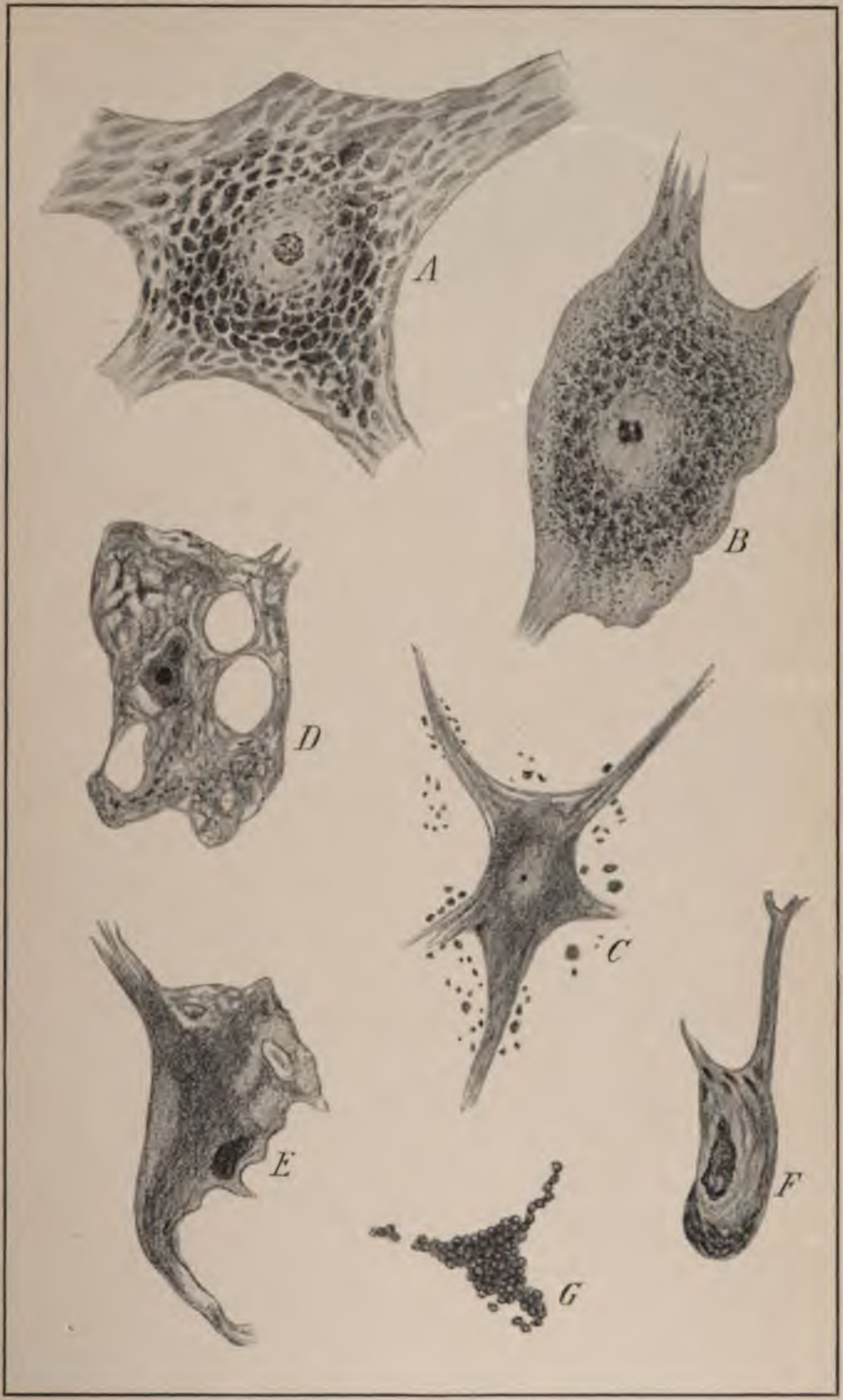
[1] Arch. of Neurology of the London County Asylums, 1899, vol. i. p. 185.

PLATE III.



Neurones in Various Stages of Degeneration.
(Schmaus and Sacki.)

A. Chromatin bodies less distinct in periphery; outline of nucleus indistinct. *B.* Chromatin changed into granules which fill the reticulum; cell body contracting. C. Entire body contracted and *filled with dust-like débris.* *D.* Body granular and containing vacuoles. E, F, G. *Final stages of degeneration.*

This is a process of hydration, and is expressed by the following equation:

$$\underset{\text{(lecithin)}}{C_{44}H_{90}NPO} + \underset{\text{(water)}}{3H_2O} = \underset{\text{(stearic acid)}}{2C_{18}H_{36}O_2} + \underset{\substack{\text{(glycero-}\\\text{phos. acid)}}}{C_3H_9PO_6} + \underset{\text{(choline)}}{C_5H_{15}NO_2}$$

Parenchymatous degeneration may be attended by or may be due to inflammation in the connective tissue or in the neuroglia which everywhere surrounds and supports the neurones. Such inflammation is termed interstitial, and the mechanical compression exerted upon the neurones by an increase in the supporting substance may cause serious degeneration in the neurone. Again, in those forms of inflammation in which we have an emigration of leucocytes, we can often see these bodies invading the neurone body and apparently destroying its tissue. It has also been shown that bacteria may find lodgement within the neurone body or in its branches, causing irritation and destruction; and it is not unlikely that in some cases the invasion of the neurone by leucocytes is really an attempt to remove these bacteria by phagocytosis. In Plate III. these changes are shown.

All these pathological processes will be more fully studied in connection with the various diseases to be considered.

II. THE SYMPATHETIC NERVOUS SYSTEM.

The sympathetic nervous system is the second division of the nervous system. Though closely connected with the cerebro-spinal system by the nerves, it appears to differ from it in its structure and to be independent of it in its function. It consists of a series of masses of gray matter enclosed in capsules termed ganglia, made up of spherical cell bodies and joined to each other and to the so-called involuntary organs of the body—the heart, bloodvessels, and lymphatics; the lungs; the digestive, hematogenetic, secretory, and reproductive systems—by nerves of a peculiar type whose axones lack insulating material or medullary sheaths, and hence are gray instead of white.

The sympathetic system is subdivided into:

1. Two great cords containing ganglia, which lie on the sides of the vertebral column and are connected with the spinal cord on one side and with the plexuses on the other.
2. Three prevertebral plexuses, the cardiac, solar, and hypogastric, which are masses of ganglia connected with the viscera.
3. Many peripheral plexuses connected with individual organs.
4. Terminal monocellular ganglia scattered through the viscera.
5. Sympathetic nerve fibres which join these plexuses and ganglia together and also establish their connection with the spinal cord and brain.

Many groups of cells lying in the central region of the spinal cord and in the lateral part of the central gray matter receive and give off nerve fibres which pass by way of the nerve roots to the spinal nerves, and thence to the sympathetic cords and ganglia.[1] These nerves are both sensory and motor; hence it is probable that some part of the activity of the sympathetic system is controlled by the spinal cord.

The connection of the sympathetic system with the brain is made by the great vagus or pneumogastric nerve.

The action of the sympathetic system in regulating the vegetative functions and the acts of the heart, bloodvessels, and lungs is usually automatic and unconscious; but this activity may be reflected upon consciousness in an indefinite manner and cause changes in the emotional state and in the general feeling of comfort. Head, who has made a careful study of this subject,[2] affirms that exaltation or depression of spirits, hallucinations, a state of suspicion, and a change of character may be induced by the reflected pains of visceral disease. He ascribes many of the symptoms which we term hysterical to such sources. It is also a fact that unusual states of consciousness, expectant attention, and conditions of the mind and brain induced by hypnotic suggestion may in turn produce physical changes in the vascular and vegetative organs. These can be explained only by admitting a control by the brain of these organs through sympathetic channels, but in an unconscious and involuntary manner.

Very little is actually known concerning the diseases of the sympathetic system, and nothing is known of its pathology. It is not improbable that some of the functional nervous affections may be traced to its derangement, especially those in which the vascular system is manifestly affected, but so few definite facts are as yet at our disposal that diseases of the sympathetic system are not included in this volume.

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[1] Onuf and Collins. Archives of Neurology and Psychopathology, 1900, vol. iii., No. 1.

[2] The Goulstonian Lectures for 1901. Brain, 1901, vol. xxiv. p. 345.

CHAPTER II.

THE NERVES AND THEIR DISEASES.

The Structure of Nerves. The Histology of a Nerve; Axis Cylinder; Myelin; Sheath of Schwann. Endoneurium. Perineurium. Injuries of Nerves. Degeneration of Nerves. Regeneration of Nerves. The Pathology of Neuritis. Parenchymatous Neuritis. Interstitial Neuritis. Segmental Neuritis.

THE HISTOLOGY OF A NERVE FIBRE.

When a nerve trunk is dissected, the connective-tissue sheath or perineurium enclosing its fibres torn away, and the individual fibres set free by tearing the finer connective-tissue strands or endoneurium which bind them together, it is possible to distinguish certain parts by means of appropriate methods of staining.

There is, first, the axone or axis cylinder (Fig. 9). This is made up of a number of primitive fibrils longitudinally arranged and continuous throughout the length of the nerve. The fibrils are cemented together by a substance which appears finely granular. Each axone comes from a single nerve cell; the individual fibrils come from the cell body and from its dendrites. The fibrils pass together in the axone to the periphery and there terminate, the individual fibrils branching out in various directions, and joining with other fibrils from other axones to form a fine plexus within the organ to which the nerve as a whole has gone. It has been thought possible to trace individual fibrils of the plexus into individual epithelial cells in various internal organs and in the skin. Other axones can be traced directly to terminal organs, such as the terminal plates upon the muscle and the terminal bulbs and corpuscles in the skin. In these no division or branching of the fibrillary constituents of the axis cylinder has been discovered. Their termination in each of the organs of special sense is still different. The diameter of an axis cylinder varies from 2.6μ to 7.5μ. It is no longer believed that the longest fibres have the greatest diameter.

There is, secondly, *the myelin sheath* surrounding the axis cylinder. (Fig. 9.) This is not a continuous tube, but consists of a series of short tubes or interannular segments placed end to end. These segments vary in length from 0.1 mm. to 1 mm. and are longer on thick fibres. The point of junction of two adjacent segments is indicated by a constriction in the contour of the nerve fibre, the node of Ranvier, and if the fibre be stained with osmic acid the myelin will be seen to be deficient at these nodes. (Fig. 6.) If, by any means, the nerve fibre be broken the myelin in any segment will run out and collect in drops, showing that it is a semifluid substance of fatty nature.

The myelin sheath is not a necessary constituent of all nerve fibres, for the majority of the nerves of the sympathetic system are devoid of such sheaths. Nor does the myelin sheath of the nerves of the cerebro-spinal system extend from end to end of the axone. For the axone first receives its sheath at some little distance from the cell from which it issues; and at its termination, where it breaks up into branching fibrils, the myelin envelope ceases. Throughout the course of the nerve, however, the myelin sheath is present in the cerebro-spinal nerves, forming a protecting envelope and probably acting as an insulating and nutrient substance as well.

FIG. 9.

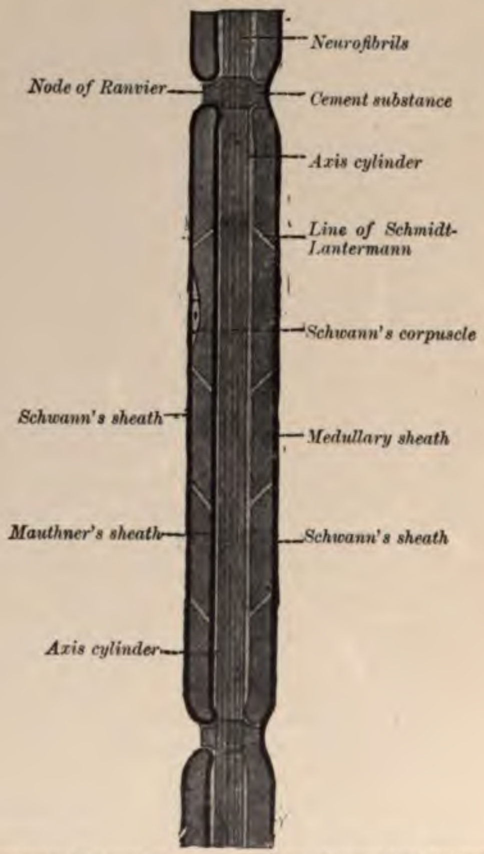


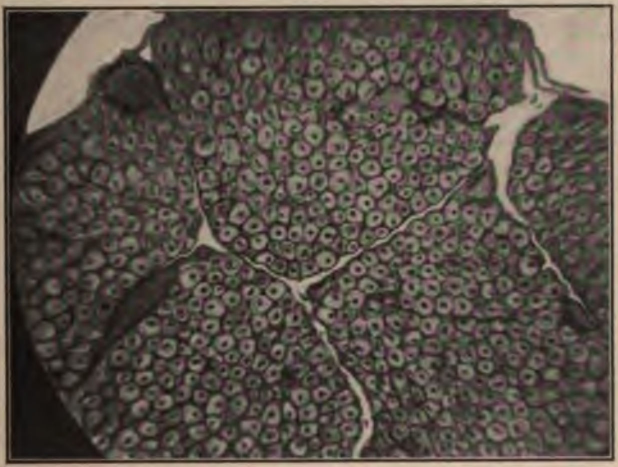
Diagram of the structure of a medullated nerve fibre, showing two different views concerning the relations of the sheaths of Mauthner and of Schwann. Compare the right and left sides. (Symonowicz.)

There is, thirdly, the sheath of Schwann—a connective-tissue membrane surrounding the myelin sheath. (Fig. 7.) This, like the last, develops in segments which become cemented to one another at the points of constriction of the fibre; but finally the continuity between adjacent segments becomes complete, so that in an adult fibre the sheath is continuous over the node of Ranvier, and here the sheath of Schwann is the only covering of the axis cylinder. On the inner side of this sheath, and half-way between two nodes, a nucleus is found. The sheath of Schwann extends almost to the terminal filaments of the axone, covering them for a little distance after they have lost the myelin sheath. By appropriate staining fine lines may be shown passing between the outer and inner layers of protoplasm through the myelin sheath; the so-called incisures of Schmitt-Lantermann. These have been considered little trabeculæ of the protoplasm within the segment, in the midst of which the myelin lies. Recent staining methods seem to indicate that they belong to the sheath of Schwann and are connective tissue. The layer of protoplasm lying against the axis cylinder is the layer which was formerly described as the sheath of Mauthner. Some authorities consider that a layer of connective tissue similar to the sheath of Schwann surrounds the axis cylinder, but this is still uncertain. Two views of its relations are shown in Fig. 9.

Thus the nerve fibre consists of a central conducting strand surrounded and insulated by a series of cylinders placed end to end and joined to one another.

Nerves. Individual nerve fibres are associated in bundles held together by fine connective-tissue cells whose nuclei in a carmine or fuchsin-stained preparation can be seen lying always adjacent to, but outside of, the sheath of Schwann. This has been called the

Fig. 10.



Cross-section of a nerve, showing five bundles of nerve fibres.

endoneurium, while the connective-tissue sheath surrounding the entire bundle is named the perineurium. The connective tissue holding numerous bundles together is called the epineurium, and this forms the firm, glistening sheath of the nerve. Capillary vessels with free anastomoses run within the nerve, their walls lying adjacent to the individual fibres, and thus affording a perfect nutrition. Lymph spaces also have been demonstrated within the nerve sheath, but not among the fibres. That the interfibrillary spaces of the endoneurium, however, open into these perivascular lymph spaces is probable from the fact that they do so in other organs. While it is evident that the nutrition of the axis cylinder is derived from the circulatory fluids, it is probable that it is only at the nodes of Ranvier that the absorption takes place, since elsewhere the myelin sheath interferes with osmosis. Thus, if the nerve be put in nitrate of silver, it is only opposite the nodes that the axis cylinder becomes stained. There is no histological difference between motor and sensory nerve fibres.

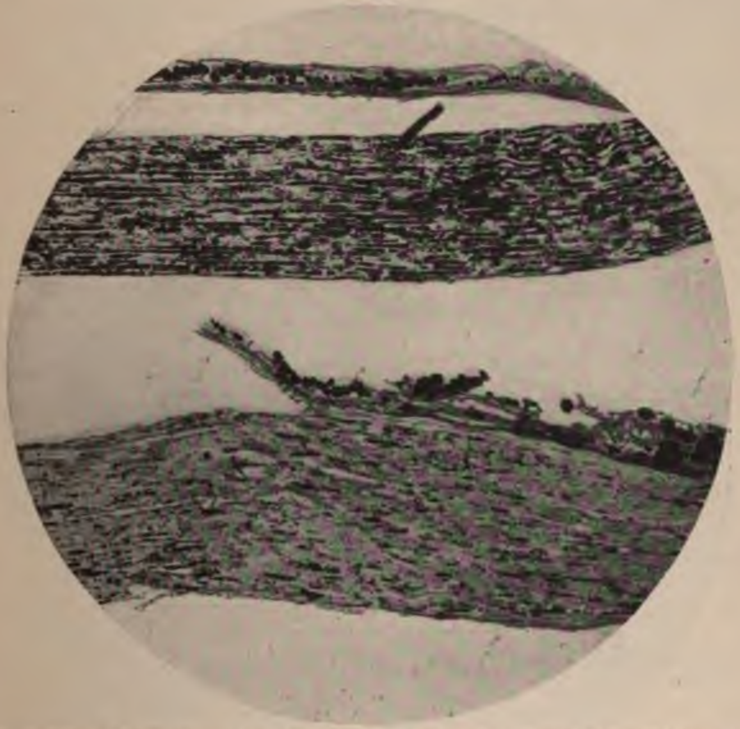
INJURIES OF NERVES.

Nerves are frequently injured, being exposed in their long course. The pathological effects of such injury and the symptoms produced are somewhat different from those due to neuritis. Hence they require a separate consideration. The pathological effects of injuries to nerves have been ascertained very largely from experimental division in animals, but there is every reason to believe that the process in man is identical with that in animals. These effects are still a matter of controversy, different observers having seen different appearances.

After division of a nerve trunk a process of degeneration sets in at the point of injury and involves a small portion of the central end, and the entire peripheral part of the nerve, from the seat of injury onward. This process may be more or less complete, and may or may not be followed by a second process of regeneration in the injured nerve. It is necessary to distinguish between the degenerative and regenerative processes.

The Process of Degeneration. When a nerve is compressed by a ligature or forceps without sufficient force to rupture the sheath of Schwann, the myelin is driven away from the point of pressure in both directions and the axis cylinder is disintegrated and mingled with it. It might be supposed that the nodes of Ranvier would prevent such a driving back of the myelin, but they seem to offer but feeble resistance, so that the entire fibre on either side of the compressed spot is bulged out for some little distance, the sheath of Schwann between the distended portions being left empty or containing only a little granular débris. If the sheath of Schwann is ruptured or is cut through the myelin exudes in little drops, which are mingled with the débris of the axis cylinder. In a short time changes of a degenerative character are observed in the nerve on both sides of the point of compression or division. Those on the central side are limited to the immediate neighborhood of the injured spot, and, according to Ranvier,[1] do not affect the nerve for a distance greater than a centimetre from the point of compression. Recent investigators, however, find certain minor grades of degeneration throughout its entire length up to the neurone body. On the peripheral side of the point of pressure the degeneration is complete, involving the entire nerve down to its finest terminations. The process begins at once in the entire length of the nerve. The first change noticed is a breaking up of the myelin sheath into segments, and then into smaller masses and drops (Fig. 11), which finally undergo further disintegration, either by a fatty or albuminoid degeneration or by a process of saponification, until a finely granular mass alone remains. Hence the contour of the fibre becomes irregular, the sheath of Schwann bulging at places with the fatty mass and at other places being collapsed and empty. Tizzoni[2] states that

FIG. 11.



Degeneration of a nerve seven days after injury. The upper specimen is a normal nerve; the lower is degenerated. Osmic acid stain.

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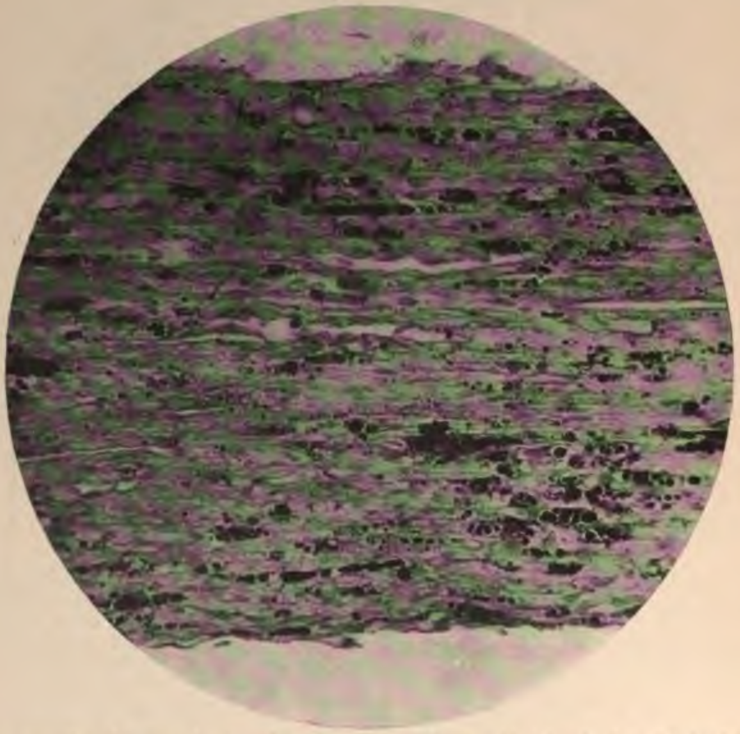
[1] Leçons sur l'histologie du systéme nerveux, tome i. p. 115.
[2] Arch. f. pathol. Anat., xviii., 1880.

this process is partly due to the activity of migratory white blood cells, a view which Ranvier supports and which Neumann[1] and Mayer[2] deny. Rosenheim[3] describes certain cells which he calls connective-tissue cells, that take an active part in all processes of nerve inflammation, and it is possible that these are the bodies that Tizzoni considered leucocytes. Ranvier holds that the segmentation of the myelin is due to the increase of the protoplasm about the nucleus of the segment, and that it is this protoplasm that replaces the myelin. Neumann holds that the granular mass resulting from the disintegration is not protoplasmic, but is a débris capable of acting as the basis for processes of regeneration after undergoing a chemical change. All authorities admit that the granular mass may be gradually absorbed, leaving the sheath of Schwann collapsed and empty, or containing only granules of débris. (Fig. 12.)

FIG. 12.



Degeneration of a nerve fourteen days after injury. Osmic acid stains the fatty débris of the medullary sheath black.

As the myelin undergoes these changes the axis cylinder usually becomes involved. Some authors, it is true, believe that it remains

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[1] Zeitsch. f. Heilkunde, ii., 1880. [2] Deut. Zeitsch. f. Chirurgie, xviii. u. xix., 1883.
[3] Centralbl. f. d. med. Wissen., 1878, Nr. 13.

intact and that, although deprived of its function, it is capable of resuming that function at any time when regeneration of the myelin sheath has taken place. Such authorities as Weir Mitchell[1] and Wolberg[2] have given their approval to this view. It is probably true for the very mild cases, but not for severe ones. Ranvier believes that the protoplasm of the nucleus attacks and destroys the axis cylinder as well as the myelin. Neumann and Mayer hold that it is split up into segments like the myelin, becomes mingled with it, and undergoes the same process of chemical change and absorption.

The sheath of Schwann also takes part in the process of degeneration. When that process has fairly begun numerous nuclei are observed lining this sheath in each interannular segment. They may have come by a process of segmentation from the original nucleus of the segment, as Ranvier holds. But Neumann and Mayer have shown that they appear as early at the extremities of the segment as they do in the vicinity of the nucleus, and therefore consider them a new formation originating in the granular or protoplasmic mass. Tizzoni thinks them emigrated corpuscles; while Rosenheim holds that they come from the connective-tissue cells along the sheath, which divide and multiply and show powers of emigration as soon as the process of degeneration begins. When the granular mass is absorbed these nuclei remain scattered along the sheath of Schwann; and it has been suggested that, when in an empty sheath a new axis cylinder appears, it owes its existence to these nuclei which arrange themselves in a line and develop into the new fibre (Wolberg). This view, however, has not met with general acceptance, though recently urged by Ballance and Stewart.[3] If no regeneration occurs they disappear gradually, and then the only relic of the former nerve fibre is the empty, collapsed sheath of Schwann, which remains as a connective-tissue strand

The increase of nuclei and connective-tissue fibrils in the endoneurium and perineurium which accompanies the process of degeneration, aids in the transformation of the nerve into a band of connective tissue. (Fig. 13.)

The degeneration that affects the nerve is continued to the terminal plates upon the muscle. These are changed into masses of granules and are finally absorbed, connective-tissue plates being left.[4] Whether any changes occur in the sensory terminal organs, such as the tactile corpuscles or terminal bulbs, has never been ascertained. Those who believe that the individual axis-cylinder fibrils terminate in the epithelium of the skin cite the trophic changes that often occur on the surface as evidence that this covering of the body shares in the nerve changes. The process of degeneration is complete in about two weeks after the injury.

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[1] Injuries of Nerves, 1872.

[2] Arch. f. Psych., Bd. xvii. 820.

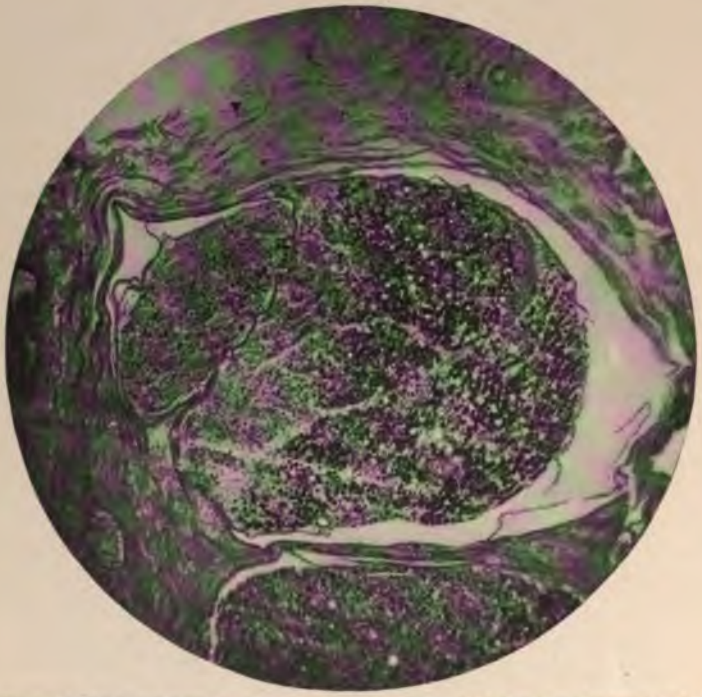
[3] The Healing of Nerves. MacMillan Co., 1902.

[4] Gessler. Die motorische Endplatte und ihre Bedeutung für die periphere Lähmung. Leipzig, 1885.

The cut end of the divided nerve becomes swollen into a bulbous extremity by a growth of connective tissue and by the development of fine nerve fibres in process of regeneration. This forms a very sensitive scar.

Whether a true union of the divided ends ever occurs is still a matter of dispute. The majority of authorities, following Ranvier, affirm that while a primary coaptation of the ends by an exudate or nerve callus, which is secondarily transformed into connective tissue, may occur and hold the ends in position, no true primary union of nerve fibres is possible, and under all circumstances the degenerative process already described goes on to completion. Gluck,[1] however, claims to have observed an actual union of the two ends, with re-establishment of function, at a time too early to have admitted the occurrences of degeneration and regeneration when less than one centimetre of the nerve is removed; and Wolberg and Bowlby,[2] approaching the subject from the surgical side and considering the results of nerve suture, incline to the same view. All surgeons

FIG. 13.



Cross-section of an ulnar nerve in a state of degeneration. The bundles of nerves on the left side are completely degenerated.

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[1] Virchow's Arch., 1878. Report of Surgical Congress at Berlin, 1880.

[2] Bowlby. Injuries of Nerves, 1896.

believe that function may be rapidly resumed after suture of freshly divided nerves, and rapid restoration of sensation, absence of wasting, and retention of muscular contractility may be taken, according to Bowlby, as certain proof of restoration of continuity of a divided nerve. It seems probable that under different circumstances different processes occur. It is undoubtedly true that in some cases the degenerative process, so graphically pictured by Ranvier goes on, from the beginning segmentation of the myelin down to the final result in the connective-tissue strand, the relic of the empty sheath of Schwann; while, in other cases of a less serious nature, the destruction is less complete and there remains a fibre consisting of a sheath of Schwann, containing a granular mass which may be either an axis cylinder or a mass capable of developing into an axis cylinder under favorable circumstances. If this is the case we can affirm that brilliant surgical successes, with rapid restoration of nerve function after suture, are possible when only partial degeneration is present, but are impossible when total and extensive destruction of the nerve fibre has occurred.

The Process of Regeneration. After the process of degeneration has gone on for some time in the nerve fibre it ceases, and the process of regeneration begins. This may commence about two weeks after an injury or experimental section. With regard to the method of this process two divergent views are held. Ranvier and his followers affirm that the new nerve is wholly a product of the central end of the injured nerve, growing out from it and making its way along the track of the peripheral end, which takes no active part in the process. Neumann and Mayer, and more recently Ballance and Stewart, have affirmed, on the contrary, that the regeneration goes on in the peripheral end of the cut nerve, segment by segment being formed successively or simultaneously, the new nerve being built up by the union of each distal segment with the one lying centrally to it, until the process is complete. These various views demand a more exact statement.

Ranvier describes several ways in which the new fibres issue from the central end. He has seen the central end of an individual nerve fibre become hypertrophied, and from this swollen part a single new fibre start out already medullated and grow onward into the old sheath, which it follows down until it reaches its end. He has also seen a single axis cylinder grow out and then divide into two, or even more, axis cylinders, each of which develops into a complete medullated nerve. Such a division of one axone into several is wholly at variance with the neurone theory and is questionable on that account. By the aid of the nerve callus or cicatricial tissue, that usually joins the central end with the degenerated peripheral end, the new fibres are directed outward toward the periphery. And when they reach the peripheral end of the cut nerve they insinuate themselves into the old remaining sheaths of Schwann or between those sheaths, and grow on and outward until at last they reach the termination of the peripheral end, and the regeneration is complete. The terminal plates upon the muscles are renewed by a reproduction of protoplasm in the plate. The process thus described is in accordance with the process of original development of nerve fibres from nerve cells in embryonal life.

The description given by Neumann and Mayer is very different. It will be remembered that they describe, as a result of the process of degeneration, a band of fibres each consisting of a sheath of Schwann containing a granular mass. They hold that the process of regeneration begins in this mass. Within it they have seen a narrow band of fine homogeneous substance appear, which has the structure of a rudimentary axis cylinder. This does not fill the sheath of Schwann and is often pressed aside by the nuclei which lie in that sheath. It is not at first continuous with the end of the old axis cylinder, remaining in the central part of the compressed or divided segment, but as it increases in definite structure it approaches this old axis cylinder, and finally unites with it. At the point of union a ring of Ranvier is formed. As this axis cylinder develops a substance is gradually formed around it, which is stained by osmic acid. This increases in thickness as the protoplasmic mass and the nuclei diminish, until it finally forms a new myelin sheath. The new myelin sheath is never continuous with the old one in the central end of the nerve, since it is separated from that by the ring of Ranvier; but often at first the old sheath seems to bulge out and encircle the new sheath, though this appearance is never permanent. At the point of union of the new fibre with the old one nuclei are often found; but these, like the others, gradually disappear. Lastly, a new sheath of Schwann is produced around the new myelin sheath and within the old sheath of Schwann. It presses aside the old sheath, together with such masses of protoplasm, drops of myelin and nuclei as may remain, leaving them thus wholly outside of the new-made fibre, so that they coalesce with and make part of the endoneurium. The new sheath of Schwann has but one nucleus in each segment and presents the nodes of Ranvier at regular intervals.

Neumann has shown that this process goes on in every individual segment of the nerve sheath, so that in segment by segment, proceeding toward the periphery, the regenerative changes occur, and as each segment approaches completion it joins itself to the preceding one, until finally the nerve is re-established in its entire length. As the degenerative process begins in the segment nearest to the point of compression, so does the process of regeneration, and in some nerves the two processes may be seen going on together, the segments near the seat of injury being renewed, while those at the periphery are still in a process of degeneration. The new fibres do not grow out from the old ones, as Ranvier describes in the regeneration of cut fibres, but protoplasm, with specific developmental properties, forms and differentiates the elements of the new fibre and then unites it to the old one. The new fibres are at first somewhat smaller in calibre than the old ones, but they gradually attain a normal size, and then the process may be said to be completed.

While Neumann would have the new fibres develop from the granular mass remaining in the old sheath of Schwann, Gunther, Hjelt, Wolberg, Weir Mitchell, and Ballance and Stewart agree that they may originate from the nuclei of the old sheath, or even from connective-tissue cells and neurilemma nuclei remaining in the connective-tissue strand. The most important evidence of such regeneration is offered by Bowlby.[1] In three cases of division of nerves in which union was attempted by operation some months after the injury, he found regenerating nerves in the peripheral portion. There were new fine fibres much smaller than natural, and in some the myelin sheath was scarcely perceptible. In some, however, the myelin sheath was fully developed, including the nodes of Ranvier. The origin of these new fibres was clearly from nuclei which seemed to be identical with the nuclei of the sheath of Schwann. These had arranged themselves in bundles with their long axis parallel to that of the nerve trunk. Then the nuclei had elongated and finally been transformed into fibres around which subsequently a myelin sheath was formed. A similar process has been fully described by Ballance and Stewart.

It is evident, therefore, that the process of regeneration varies in different conditions according to the exact stage of degeneration reached before it begins. If the final product of degeneration is a band of simple connective tissue, it seems probable that the nerve fibrewill have to grow into it from a central origin, as in its original development in fœtal life. If, however the connective-tissue cells recently discovered are neuroplastic cells and have the power of producing new nerves, just as cells of periosteum may produce a new bone, and if, when degeneration ceases, there remains a sheath of Schwann containing a granular protoplasmic mass, it is not at all improbable that that mass may be differentiated into an axis cylinder and a medullary sheath and joined to the old nerve fibre—a process which has its analogy in the medullation of nerves in the embryonal state. If we admit, with Wolberg, that in some cases the axis cylinder is not destroyed, the formation of new myelin is a rapid matter. That some such process as the one described by Bowlby and Stewart must occur in many cases is certain, when the rapid recovery after minor injuries is considered and when the results of nerve suture are taken into account, for in both these conditions the return of function occurs long before a new nerve fibre starting out from the old one could have reached the periphery.

It is affirmed by Mayer that individual nerve fibres in normal nerves are constantly undergoing these processes of degeneration and regeneration, either because the necessary renewal of worn-out tissue takes place in this manner, or because slight injuries from pressure or overstrain are sufficient to start up degeneration in single fibres. Such changes are more apparent in old age than in youth, and in cachectic conditions than in healthy states. In all persons dying of infectious diseases they can be found well marked.

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[1] Injuries of Nerves, page 25.

The process of degeneration in the nerves consequent upon the destruction of the ganglion cells from which they arise—the so-called Wallerian degeneration—which is best seen in cases of anterior poliomyelitis, differs in no respect from that ensuing upon compression or division.

NEURITIS.

A nerve may be inflamed in a short portion of its course—localized neuritis; or it may be affected at many different parts—disseminated neuritis; or it may be diseased in its entire length—general neuritis. The process may be described as ascending or descending, according to the direction in which the disease makes progress. Even when the lesion is a strictly local one, limited to a short portion of a nerve, extensive secondary changes occur from the part affected outward; and, as these may involve the entire length of the nerve, regeneration and repair may require a much longer time than is taken by the healing of the original lesion. General constitutional states may produce a simultaneous neuritis in many nerves—multiple neuritis—and as this condition develops usually in the distal parts of the nerves, it is often termed peripheral neuritis. Neuritis may also occur secondarily to inflammatory changes in other parts, as with periostitis or abscesses. Syphilitic deposits in the nerves, tubercles in the nerves, cancer or other neoplasms along the nerves may cause a proliferation of the connective-tissue elements or a true diffuse inflammation.

Pathology. A nerve which is inflamed is red and swollen, is lacking in its natural surface lustre, and is no longer firm and smooth to the touch. Its vessels are congested and there may be hemorrhages within its sheath. If the process has been in progress for some time there may be bulbous swellings on the nerve the result of connective-tissue infiltration, or the nerve may be markedly atrophied. Such bulbous thickenings are very common after injuries and always occur after a division of a nerve on the central end. A section of the nerve viewed by the microscope shows a distention of the vessels, an infiltration of endoneurium with small cells, and a thickening of the connective-tissue elements.

The microscopic changes are similar to those produced by injuries of the nerves. Yet it is possible to distinguish between cases in which the lesions are chiefly limited to the axone and medullary sheath—the parenchymatous form of neuritis, and cases in which the lesion affects more especially the connective tissue about the fine fibres and the endoneurium and perineurium—the interstitial form of neuritis. In the latter the bloodvessels and lymphatics take a more active part in the inflammatory changes.

In parenchymatous neuritis at the outset the myelin sheath appears slightly swollen, is less homogeneous, and, from a difference of refractive power, is less translucent. It then becomes split up into segments of different length and form, the segmentation occurring preferably at the incisures of Schmitt, while the incisures at other parts disappear. Between these segments of myelin a finely granular protoplasm is seen in which new nuclei are found. In some fibres the axis cylinder may still be preserved. In others it is broken at the same places as the myelin.

At the next stage of the process the changes are more marked. The myelin is now reduced to a series of small globules surrounded everywhere by granular protoplasm, and in this protoplasm the nuclei are now very numerous. The axone cannot be distinguished in the mass, as a rule, but occasionally a fine line is seen passing through the mass, which may be a remaining axone. (See Fig. 12.)

The succeeding stage presents a different picture. While up to this time the size of the nerve fibre has remained about normal and uniform, it is now seen to vary. At places the fibre is still wide and filled with a granular mass; at other places it is narrow, the mass having disappeared, leaving either a collapsed sheath or a sheath containing only nuclei here and there. In a few such narrow fibres there seems to be an axone lying directly within the sheath of Schwann, and occasionally separated from it at various places by nuclei; but, as a rule, no trace of the axone remains. As any single fibre may show constrictions at some places, dilatations at others, the variation in its calibre is the most striking feature of this stage. In the terminal stage the calibre is uniform again, but is now everywhere reduced. The sheath of Schwann is empty or contains only a little granular substance and the nuclei are now less numerous than before. There is, in fact, only an atrophied tube with none of its original contents. These tubes lying side by side are folded and undulating, and appear like a strand of connective tissue.

These various stages of parenchymatous inflammation are to be seen in different fibres in the same specimen. They are present not only at the seat of inflammation, but they are present from this point onward to the end of the nerve, constituting the change known as secondary degeneration. Their appearance is identical with that observed in the course of degeneration of a nerve after compression or after destruction of the neurone body in the spinal cord, as in anterior poliomyelitis. This had led such an accurate observer as Erb[1] to advance the hypothesis that in cases of multiple neuritis due to toxic agents some slight changes in cells in the spinal cord, not visible to the microscope, are present primarily, and that the changes in the nerves are secondary, the nutrient power of the cell being incapable of supporting the entire axone, which thus shows changes in its most distal part. It has lately been shown by means of the Nissl stain that after any nerve lesion in the periphery a change occurs in the entire length of the axone and also in the central cell body, of which that nerve is the axone. (See Plate I., *G.*) This change is a degeneration; but at the end of a month it becomes stationary, and then gradually the cell body regains its original appearance, even though the axone may remain degenerated. Such cell change, therefore, is not necessarily the primary condition in neuritis. Strümpell urges that parenchymatous neuritis has its parallel in other parenchymatous inflammations, and therefore does not need to be traced to any primary affection in the cells; and hence Erb's hypothesis has not met with general acceptation.

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[1] Neurol. Centralbl., 1883, p. 481.

Interstitial neuritis presents a different appearance. In this condition inspection shows the nerve to be congested, swollen, thicker than normal, and lacking in lustre, or to be yellow and irregularly swollen by the accumulation of pus and serum, or to be reduced to a mere connective-tissue strand. Upon teasing the nerve it is at once evident from its brittleness, that individual fibres are lacking in continuity and are changed in structure; and if it is examined under the microscope the exudation of serum and of inflammatory corpuscles, the great increase in the number of connective-tissue nuclei, the distended condition of the vessels, as well as the various appearances characteristic of nerve degeneration, are clearly seen. The thickening and hyperplasia of endoneurium and perineurium are well marked in the older cases. Here the inflammation is originally an interstitial inflammation, though later it becomes a diffuse one. It is possible that the degenerative processes in the nerves may have been due to the compression by the products of inflammation exuded within the nerve sheath. In one or two cases where the patient died early in the disease the nerve fibres which lay near the vessels were affected to a greater degree than those lying deeper, and from this fact it was concluded that their degeneration was secondary. In other cases, however, all the fibres in a bundle were equally involved. It is probable that in some cases the inflammation is diffuse from the outset, parenchyma and interstitial tissue being affected simultaneously. One marked feature in these cases is the large amount of fatty deposit found in the altered nerves, as shown by the Marchi stain. This is to be ascribed to the fact that myelin in undergoing degeneration breaks down into globules and undergoes a fatty change. It is itself allied to fat, and making up, as it does, a large part of the bulk of the fibre, it would be noticeable in the product of degeneration if it were not absorbed. Sometimes the absorption seems to be interfered with by the vascular condition, and hence the residual amount of fat is increased. (See Fig. 11.)

One additional pathological form must be mentioned, since it has been described by such a careful observer as Gombault.[1] It is the

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[1] Archives de Phys., 1873, p. 592; also Arch. de Neurol, i. 1.

so-called segmental periaxillary neuritis. In toxic neuritis from lead and alcohol poisoning, Gombault found that the degenerative process was not uniform in the entire length of the nerve fibre. On the contrary, entirely normal segments alternated with the degenerated segments in the nerve. Mayer has noticed a somewhat similar condition, and it has been described as occurring in senility. The changes already described take place in the myelin sheath of the affected segment, even to its entire absorption, leaving the axis cylinder in the sheath of Schwann; but, as the adjacent segments are not involved, regeneration is thought to be more easily accomplished. Pitres and Vaillard[1] have noticed a somewhat similar condition in the neuritis occurring after diphtheria, although in their case the axis cylinder as well as the myelin sheath was totally destroyed in many segments.

Occasionally a simple atrophy of nerve fibre has been observed; a mere gradual reduction of all the elements without any degenerative process.

While it is, of course, impossible to describe definitely the process of regeneration that goes on after neuritis, there is no reason to suppose that it differs in any way from that observed in experimental lesions. The time required for the completion of the process will depend upon the severity and extent of the degeneration. When that is slight the recovery may be rapid, cases having been reported where a total restoration of function took place in two months. As a rule, however, it is a slow process. The large majority of the cases require over six months for the complete regeneration, and in not a few cases from ten to sixteen months elapse before the condition of the nerves is proved to be normal by the total disappearance of all symptoms.

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[1] De la névrite segmentaire, Arch. de Neurol, xi. 337.

CHAPTER III.

INJURIES AND WOUNDS OF NERVES AND NEURITIS.

Symptoms of Injuries to Nerves and of Neuritis. Diagnosis. Treatment of Nerve Injuries and of Neuritis. Neuromata.

Symptoms. A very slight pressure upon a nerve, of short duration, may cause disagreeable tingling and numbness of the skin, which is felt in the distribution of the sensory filaments of the nerve. This is an experience common to everyone when from pressure on the sciatic nerve the foot is said to be asleep. Such sensations of numbness often occur in the arm and hand, or in the foot and leg at night during sleep and wake the patient, who may be alarmed, and, from a recurrence of the symptoms, anticipate paralysis, especially if the numb sensation is temporarily attended by awkward movements or by a feeling of weakness. Inasmuch as the relief of the pressure is followed by a rapid recovery from the symptoms such sensations are of no importance. They are due to a slight impairment of nutrition in the nerve probably consequent upon a venous congestion, for a similar set of sensations can be caused by tying a string tightly about the finger or wrist and causing a stasis of venous blood. Some persons seem to be especially subject to such symptoms. Such a condition does not ordinarily amount to a neuritis, but if it is long continued it may produce it, as is seen in cases of musculospiral palsy from sleeping on the arm in a state of intoxication.

A slight general numbness of all the fingers in both hands, and even of the hands and wrists as well, occasionally occurs in attacks of short duration or may become permanent. This is noticed chiefly in women whose hands are constantly immersed in hot water or who use the hands continuously for hours in any employment. It has been called *acroparæsthesia*.[1] It is due to a slight impairment of nutrition in the nerves, and if the motor as well as the sensory nerves are affected it is attended by a sense of weakness and lack of skill. Disturbances of circulation are sometimes the cause, as in numbness from pressure. These symptoms are variable in duration and may continue if the cause is not removed; but they never go on to any serious disease, and hence are of little importance. Rest from work and massage are the best remedies.

The effect of an injury or wound of a nerve or plexus of nerves is immediate pain at the point of injury, pain or disagreeable sensations, such as numbness or prickling, referred to the regions from which the sensory filaments in the injured nerve come, anæsthesia in this region to all forms of sensation, of temporary or permanent duration, occasionally attended by trophic disturbances, and paralysis of the muscles to which the motor filaments in the injured nerve pass. The paralysis is soon followed by a loss of faradic contractility in the muscle and by a loss of its mechanical excitability to percussion, and later by a progressive atrophy of the muscle.

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[1] Dana. New York Medical Record, January 20, 1891. Collins, New York Medica Record, December 3, 1901.

At the point of injury to the nerve the symptoms vary. They differ when the skin is unbroken, when the nerve is exposed to the air, and when the wound is infected. In the first case, while there may be slight tenderness to pressure there may be no spontaneous pain. Thus in a case of bilateral ulnar paralysis from pressure exerted on the nerves of the arms during a long surgical operation, where the arms hung over the sides of the operating table for an hour and their weight against the sharp edge caused the injury, the patient had no pain at all and no tenderness at the point of injury. In the common form of injury to the musculospiral nerve from pressure on the arm by the head during the heavy sleep of intoxication, the patient wakes with wrist-drop, but has no pain. In facial palsy there is rarely pain behind the ear. Thus where the injury is produced by long-continued slow pressure sufficient to destroy the nutrition of a nerve there may be no pain. On the other hand, pressure on a nerve by a tumor or pressure by a pregnant uterus on the lumbar and sacral plexus may give rise to severe pain, both local and distal. If the nerve is lacerated suddenly, as by a strain or a blow or by a fracture of a bone tearing it, there is always severe pain at the point of injury, and this pain is usually much increased if the nerve is exposed to the air in an open wound. In these cases also the subsequent connective-tissue growth and thickening about the wounded ends is tender and may cause great pain, the finer filaments becoming entangled in the scar tissue.

The pain after injury is often felt not only at the point of injury but distally in the entire peripheral distribution of the nerve. Thus in a case of ulnar nerve injury occurring with a fracture at the elbow there was pain of an agonizing kind in the inner condyle of the humerus and also severe shooting pains down the inner side of the forearm and into the hand and little and ring fingers; these pains grew worse for several days and continued for many weeks, with intense tenderness in the entire ulnar nerve distribution, any touch in this region causing agony. Ulcerations of the skin of the fingers and hand occurred later and the skin became glossy. This condition remained stationary until by operation at the elbow the nerve was released from a mass of callus, and thus was freed from pressure, when all the symptoms gradually subsided and recovery ensued.

The pain and tenderness caused by an injury of a nerve are not always limited to the distribution of that nerve, as one would expect. Thus in a case where a perforating wound of the sole of the foot had left a small piece of rubber shoe embedded in the flesh the original injury was slight, the wound healed, and for a long time no inconvenience was felt; but four years later pain and tenderness began again in the scar and extended up the leg and thigh until the entire limb became painful, hypersensitive, and useless from the intense tenderness, and even the lower spine was sensitive and painful. These symptoms all subsided rapidly when, by operation, the piece of rubber was removed. It was found to be surrounded by a little mesh of fine nerve filaments holding it like a basket. The rapid recovery showed that there was no true ascending neuritis, as had been supposed. In such cases the continued local pain causes a hypersensitive state of the central organ by a summation of impressions, and this shows itself by a general pain. In many cases of neuritis of one cord of the brachial plexus the pain in the entire arm renders it useless. The pain in neuritis is increased by pressure upon the affected nerve. It is also intensified by motion of the limb, especially if this causes a stretching of the nerve.

The anæsthesia following injury of nerves is more pronounced and extensive immediately after the injury than it is later. There appear to be fine anastomoses of the sensory filaments of nerves in the skin, and thus sensations ordinarily conveyed by one nerve may, if that nerve be injured, pass by adjacent nerves. Such anastomoses seem to be more complete in some individuals than in others, hence in two patients with similar injuries the anæsthesia may be different in extent. Thus after injury of the musculospiral nerve it is very common to find sensation in the back of the hand near the thumb perfectly restored within a week, while the wrist-drop may last some months. After a division of the median nerve above the wrist I have seen the anæsthesia progressively diminish in the hand long before the union of the ends of the severed nerve had restored the muscular power in the flexors of the fingers. After excision of one branch of the trigeminal nerve for neuralgia, sensation returns in the face in a considerable part of the region which is anæsthetic immediately after the operation; but a small area of anæsthesia is usually left as a permanent result of injury to any sensory nerve, and this includes analgesia and thermo-anæsthesia. A loss of muscular sense rarely, if ever, follows a nerve injury. This is because many different sensations combine to produce this sense, deep and superficial nerves from muscles, ligaments, and articular surfaces all conveying impressions which are co-ordinated in the spinal cord before being sent up to the brain and appreciated as muscular sense.

As regeneration occurs in the injured nerve sensations gradually return in the previously insensitive region, but at first all sensations are attended by an abnormal sense of tingling or numbness which is disagreeable. This tingling or numbness is often perceived during the entire course of the disease. It is then due to irritation in the central end of the injured nerve, the pathological irritation being referred erroneously to the region whence the irritated filaments come. Such numbness may be attended by pain of a sharp, shooting character, and in some sensory nerves, notably the trigeminal and sciatic, this pain may be the most important symptom of neuritis from pressure. Thus in a case of osteosarcoma of the pelvis, sciatic neuralgia, supposedly functional, preceded for seven months all other symptoms. In a case of osteoma of the skull near the optic chiasm trigeminal neuralgia preceded by one year the optic atrophy and other symptoms. When septic infection occurs in the injured nerve this pain is more intense than under other circumstances, and it is then always attended by a state of hyperalgesia in the affected area. Such a state is known as anæsthesia dolorosa, for pain is caused by any touch in the sensitive region, even though the touch itself is not perceived.

Trophic disturbances in the anæsthetic region occur more commonly in septic cases, but they may develop in any case. They are usually produced by neglected injuries of the insensitive region, which become infected, a slight pin prick or a scratch being capable of originating a serious trouble if not disinfected at once. Pain is nature's means of protection in health, giving warning of injuries or diseases and leading to their care; but from insensitive regions no warnings can come, and no protection is therefore given. But this is not a sufficient explanation for the occurrence of such trophic symptoms as arise in neuritis, either traumatic or spontaneous. We see peculiar bulbous eruptions, herpes, acne or eczema, extraordinary vasomotor conditions, with alternate flushing and pallor of the skin attended by intense heat and cold, a dryness and thinning of the skin, with a peculiar, smooth, shining appearance termed glossy skin, unusual sweating, which may be acid and fetid, or hardening and thickening of the skin, or abnormal growth of hair, or falling of the hair; a defective or irregular growth of the nails, which may be ridged, curved, and marked with white plaques in the region supplied by the affected nerve. Very rarely acute gangrene has been observed, chiefly in old persons. The occurrence of these symptoms has given rise to the hypothesis of trophic nerves, whose function is to regulate nutrition and repair and whose injury results in processes of disease in the derma and its adnexa. Such trophic symptoms develop only when the nerve injured is a sensory nerve. The trophic disturbances seen in cases of locomotor ataxia and syringomyelia are evidence that central as well as peripheral lesions may be attended by such symptoms; but even in these diseases it is the sensory portion of the nervous system to which the pathological change is limited. The true explanation of trophic changes is to be found not in the hypothesis of an injury to trophic nerves, but in the fact that ordinary sensory impressions are interrupted or perverted, and nature, lacking its accustomed guide to repair and misled by abnormal impressions, produces effects which are needless under the circumstances or fails to produce those which ordinarily would be required. Thus, an

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increased secretion of sweat may be a natural thing under a certain condition—*e. g,* heat—when unnatural it may result from a series of abnormal sensations of pathological origin, being received by the central organs which are incapable of distinguishing natural from artificial impulses. A bed-sore is usually produced by pressure or irritation of the skin, not perceived, and so not removed or repaired. If, by extraneous care, such pressure and irritation are avoided, as in health would occur by change of position and care of the skin, bed-sores may be entirely prevented. In a patient of my own suffering from transverse myelitis with pronounced tendency to bed-sores, the simple expedient of turning him every half hour day and night and wiping off the skin lightly each time that he was turned prevented for four years the development of any skin abrasion. I have never seen trophic skin affections occur in the course of neuritis when the parts were properly protected from injury, and slight injuries were antiseptically treated.

It must be stated, however, that other theories than the one here offered have been advanced to account for the trophic symptoms in neuritis. They are ascribed to vasomotor disturbance. Some authors believe that in the course of neuritis the vasomotor nerves are diseased and that their lesion results in an irregularity of blood supply to the part concerned. This irregularity consists of an active hyperæmia, as is proven by experimental section of the sympathetic nerve to a rabbit's ear, which causes extreme dilatation of the vessels and a rise of temperature in the part; but it has never been demonstrated that a mere condition of congestion goes on to a condition of inflammation unless other causes, chiefly septic or microbic, enter as a factor. Furthermore, it is well known that the bloodvessels are under the control of vasomotor ganglia in their walls, which regulate their calibre even after division of the fibres which connect these ganglia with the spinal cord. Thus, after division of the sympathetic in the ear of a rabbit, the congestion gradually subsides in the ear and the temperature becomes normal. Another argument against the vasomotor origin of trophic disturbances is derived from the study of cases of erythromelalgia. This is an affection first described by Weir Mitchell, characterized by a dilatation of the arteries in the extremities resulting in an extreme condition of redness and sensation of heat and pain. It is an affection of the hands and feet and occasionally involves the arms to the elbows, and the legs to the knees. After hours of extreme heat and flushing the extremities may suddenly become cold, pale, or blue and shrivelled up, presenting the appearance that the hands present after long soaking in hot water, and this alternation of distention and contraction of the bloodvessels constitutes the symptomatology of the disease. It appears to be a pure vasomotor neurosis, but it is rarely, if ever, attended by any trophic disturbances of the skin or of the nails, such as occur in neuritis, and no pathological observations are at hand to establish the hypothesis that there is a true neuritis of the vasomotor nerves. For this reason the theory of the vasomotor origin of trophic affections appears to be incredible.

The paralysis which results from traumatic neuritis is limited to the muscles supplied by the injured nerve, and is total only in those muscles which have no collateral nerve supply. It is a flaccid palsy, and if it results in any stiffness of joints or limbs or deformity soon after the injury, this is due to contraction in the healthy unopposed muscles and not to contracture in the paralyzed ones. After a long-continued paralysis, however, attended by atrophy, a shortening of the weak muscle may occur. Thus, in cases of facial palsy which do not recover, a contracture is not very rare, causing stiffness of the face. In such paralyzed muscles no mechanical or reflex motion is possible. Percussion of the muscle or of its tendon, therefore, fails to produce any response.

Electrical changes soon develop in the muscles paralyzed, usually within four or five days of the time of injury. These are of several kinds and are known as the reaction of degeneration, partial or complete. In health a muscle may be made to contract by sending a galvanic or faradic current through its nerve, or by applying either current directly to the muscle. In neuritis the nerve becomes inexcitable to any form of electricity and the muscle shows changes in its excitability. A number of different conditions have been observed. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost, and then the muscles react to a galvanic current only. They may be at first hypersensitive to galvanic stimuli, but later it may require a very strong galvanic current to produce contraction. The contraction is rarely quick, as in health. It is sluggish and vermiform. In some cases the normal polar reaction is found, and the closure of the negative pole applied to the muscle produces stronger contractions than the closure of the positive pole. This is termed a partial reaction of degeneration. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole. This is termed a complete reaction of degeneration. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle are, therefore, important symptoms of neuritis. As the disease goes on to recovery a gradual increase in the galvanic irritability occurs—a fact which is often of much aid in prognosis if careful measurements of the strength of the current used be made by the galvanometer. It is possible to record such measurements upon charts, and thus to obtain an electric curve for each muscle which is paralyzed.[1] These curves enable one to judge of the progress of the case very accurately, and when the line is advancing steadily toward the normal point, after a great deflection or after a stationary level, the prognosis is favorable.

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[1] Journal of Nervous and Mental Disease, February, 1887, vol. xiv.

It is to be remembered, however, that voluntary power always returns some time before electric reactions become normal.

The extent of the paralysis in any case is determined by the distribution of the nerve affected. Thus in facial palsy all the muscles of the face except the orbicularis oris are paralyzed. In musculospiral paralysis the extensors of the wrist and fingers are totally paralyzed and the supinator longus is partially paralyzed. In peroneal paralysis the muscles which abduct and lift the foot are useless.

The duration of the paralysis depends wholly on the question of the possibility and time of regeneration of the nerve. In case a nerve is divided and at once reunited this paralysis may disappear in a couple of weeks. In an ordinary traumatic case or after inflammation, as in facial palsy, six weeks may be the limit. In other cases a long time, six or eight months, elapses before the paralysis passes off. The further the injury from the end of the nerve, the longer the time to recovery—a fact which gives support to the hypothesis of Ranvier that the new nerve must grow out from the central end at the point of injury and find its way down the old sheath to the muscle. In musculospiral paralysis from pressure over the humerus seven months is the average duration to recovery. In case, however, some obstruction to repair exists the paralysis is permanent. Hence scar tissue which prevents the new filaments from developing must often be removed in order to effect a cure.

Spasm of the muscles supplied by the injured nerve occasionally occurs, but is a rare symptom. It is usually a reflex phenomenon due to sensory irritation and not to any pressure on the motor nerve. In a gunshot injury of the ulnar nerve cramps in the hand and extensors of the wrist have been noticed (Raymond). In the so-called occupation neuroses which some authors ascribe to neuritis, such cramps are common, but in ordinary cases of neuritis they do not occur.

While the statements of the symptoms occurring in neuritis thus far made are generally true, it is found that neuritis in different nerves produces diverse symptoms, hence a special consideration of neuritis of the different nerves is necessary.

After wounds and injuries of the nerves a condition has occasionally been seen which is known as *ascending neuritis* or *migratory neuritis*. Tender spots along the course of the nerve above the point of injury and pain in the course of the nerve as high as its root in the plexus have been observed in a few cases. This has been chiefly in patients in whom there has been an open wound at the point of primary injury and where there has been a suspicion of an ascending septic process in the nerve. The neuritis, however, is not always a continuous one from the point of injury upward, but in a few cases tender spots have been found at some distance above, without any change in the intermediate space. Hence the term migratory neuritis. The migratory form is supposed to be characteristic of septic infection, the sepsis extending along the connective-tissue sheaths of the nerve. When there is no open wound to account for this sepsis, a septic condition of internal origin, associated with obliteration or plugging of the bloodvessels and with the production of a gangrenous area, has been recorded as a cause. While cases of ascending neuritis in the continuity of the nerve have been produced experimentally, it has not been possible to produce migratory neuritis when the wound has been kept aseptic. In some cases of traumatic neuritis a very extreme condition of tenderness of the limb above the point of injury, together with inability to move the joints and a general hypersensitive state with pain in the joints, occasionally develops. This condition, however, must be considered as probably hysterical, and usually develops only in hypersensitive or neurasthenic individuals. It is not necessarily attended with a distinct localizable tenderness along the course of the nerve, though this may also be present. The number of cases of ascending neuritis recorded in the literature of the past few years is very few. I have never seen a case of true ascending neuritis, though I have seen many cases of painful affection of the extremities above the level of the neuritis that were hysterical in their nature. There are some cases which are not hysterical. In these the explanation of the condition is found in the fact, well known to psychologists, that mild irritation long continued produces in a nerve centre a hypersensitive state by what is known as a summation of impulses. In this state slight impressions are believed to be intense and local impressions become generalized and widely referred. This state may be induced by neuritis long continued and has been mistaken for an ascending neuritis.

Diagnosis. The diagnosis of traumatic neuritis usually presents no difficulty. The limitation of the pain, anæsthesia, and paralysis to the distribution of a single nerve, and the tenderness at some point along its course are characteristic of the affection at the onset, and within a week of the beginning the development of the reaction of degeneration in the muscles supplied by the nerve affected makes the diagnosis complete.

Prognosis. The prognosis in neuritis, as a rule, is a good one. We have seen that there is a spontaneous tendency to regeneration in a nerve that is injured or that has been affected by inflammation, and while this progress toward recovery is usually slow, yet eventually it becomes complete and all the functions of the nerve are restored. The only factor in preventing a recovery is the impossibility of a union between the severed ends of the nerve, or the interposition of callus, or the development of a connective-tissue scar which prevents a re-establishment of continuity; but, after such obstructions are remedied by surgical treatment, regeneration takes place, even though the obstruction may have persisted for many months. Therefore, the eventual prognosis of recovery in neuritis is good. Spontaneous recovery, unless the nerve is put in a normal condition by being freed from scar tissue, may not occur. I have seen a man, aged fifty-two years, who had suffered since the age of six years from paralysis and atrophy of the muscles controlled by the ulnar nerve. At that time his elbow was fractured and dislocated and the nerve displaced, so that it passed over the olecranon, where it could be felt. Until the age of fifty years he suffered from partial paralysis and anæsthesia, and then, from unknown cause, his symptoms, pain, numbness, and atrophy increased rapidly, and he suffered much. Complete recovery of power and sensation followed an operation that repaired the nerve, though so many years had elapsed since the injury.

Bruns[1] has recently called attention to the ultimate results in injuries of the nerves and of the plexuses, and has shown that while two-thirds of his cases of nerve injury recovered, only about one-quarter of the cases of injuries of the plexuses were cured.[2]

Treatment. In the treatment of injuries and wounds of nerves or in spontaneous neuritis the first and most important object is to secure the possibility of regeneration by establishing the continuity of the injured nerve. If the injury is from a stab wound it is important that the severed ends of the nerve should be brought together and carefully united, or if laceration has taken place, so that there is a loss of continuity, a flap can be made from both ends and these elongated nerves then united; or it is possible to insert the ends within a tube of decalcified bone to serve to direct the regenerating fibres outward toward the distal end. Even those who teach that regeneration occurs in the severed end admit that it is only rapid and perfect when such union is established. If the nerve has been lacerated by a fracture of bone or by a strain it is usually sufficient to set the fracture or to place the strained part in splints, thus securing its immobility for a time and allowing nature to effect the proper repair. Occasionally, however, when the fracture has united, or when the local swelling and pain of the strain have gone down and the bony thickening or the induration about the strained part has disappeared, the nerve will still be found to have lost its function. If after six weeks' treatment by electricity and massage it is still evident that there is no progress toward recovery, it is advisable to expose the point at which the nerve was injured. It is sometimes found that regeneration has been prevented by the growth of a connective-tissue mass, and occasionally it is found that a mass of bony callus has formed about the nerve in such a way as to keep up pressure. Thus, in a case of ulnar neuritis under my observation, produced by fracture at the elbow, the repair of the fracture and the restoration of motion in the joint were not attended by a recovery from the neuritis. It was evident, by palpation, that a bony callus still existed about the nerve at the elbow, and surgical exploration showed the nerve to be completely embedded in this mass. When this was chiselled away and a groove formed in which the nerve could lie freely, and the two ends were approximated by a flap, regeneration and recovery ensued. In a case of an infant in whom brachial neuritis from pressure above the clavicle during delivery had occurred, and a permanent paralysis of the arm had remained for seven years, surgical exploration discovered a mass of scar tissue lying upon the nerve trunk and about twice its diameter, which had prevented regeneration, and when this was removed and the ends of the nerve approximated recovery ensued. It is evident, therefore, that the first requisite in the treatment of injuries of nerves is to restore, if possible, the original position of the nerve, so that nature may produce regeneration.

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[1] L. Bruns. Neurol. Cent., Nov., 1902.

[2] Kennedy. Brit. Med. Jour., Feb. 7, 1903.

In cases of spontaneous neuritis from cold, etc., the only thing necessary is to maintain the parts in a perfectly quiet position, and, if possible, to reduce any congestion in the inflamed nerve. This may be done by the use of counter-irritants, of which the actual cautery is probably the best. Light touching with a Paquelin cautery along the course of the inflamed nerve will often give relief to intense pain and will reduce the congestion which attends spontaneous neuritis. Small mustard plasters are also of service.

Local applications of heat are often very grateful in painful conditions of neuritis; in fact are much more agreeable to the patient than cold, hence poultices or packing the affected limb in cotton-wool covered with oiled silk may be of service. In the more severe cases, where pain is frequently intense and keeps the patient awake at night, it is necessary to use medicines. The newer analgesics seem to have the power of diminishing painful sensations in peripheral nerves, and hence phenacetin, 5 grains; antipyrine, 10 grains; acetanilid, 3 grains; exalgin, 3 grains, or salophen, 10 grains, may be used every two or three hours. It is sometimes possible to obtain a better action by a combination of these, to which a small amount of codeine may be added if the pain is very persistent. The following formula is a favorite and very efficacious:

℞.—Phenacetin	gr. vj.
Acetanilid	gr. iij.
Codeine	gr. ¼.
Caffeine	gr. ij.
Sacch. lactis	gr. v.

M. Triturate. Sig.—One such powder every three hours.

Codeine is a useful remedy, but in many cases it is necessary to resort to hypodermic injections of morphine. These may be used freely and with less danger of producing a habit in neuritis than in any other affection, inasmuch as the gradual recovery will insure a diminution of the pain and the eventual cessation of the use of the drug. The tenderness of the nerve to any manipulation and the production of pain by any movement are sufficient to induce the patient to keep the part in perfect rest, which is essential to recovery. The anæsthesia in the skin within the domain of the affected nerve can be speedily reduced and in many cases entirely removed by the application of the faradic brush. One pole of the battery should be placed high up upon the limb that is affected, or upon the trunk, and the brush should be applied in the anæsthetic area. The strokes should be from the adjacent sensitive skin into the anæsthetic region, and the strength of the battery should be moderate, yet just sufficient to produce a distinct sensation of the electric current. In this way it seems as if it were possible to produce an extension of sensibility from the sensitive area into the adjacent insensitive region, and thus to open up anastomotic paths through the skin.

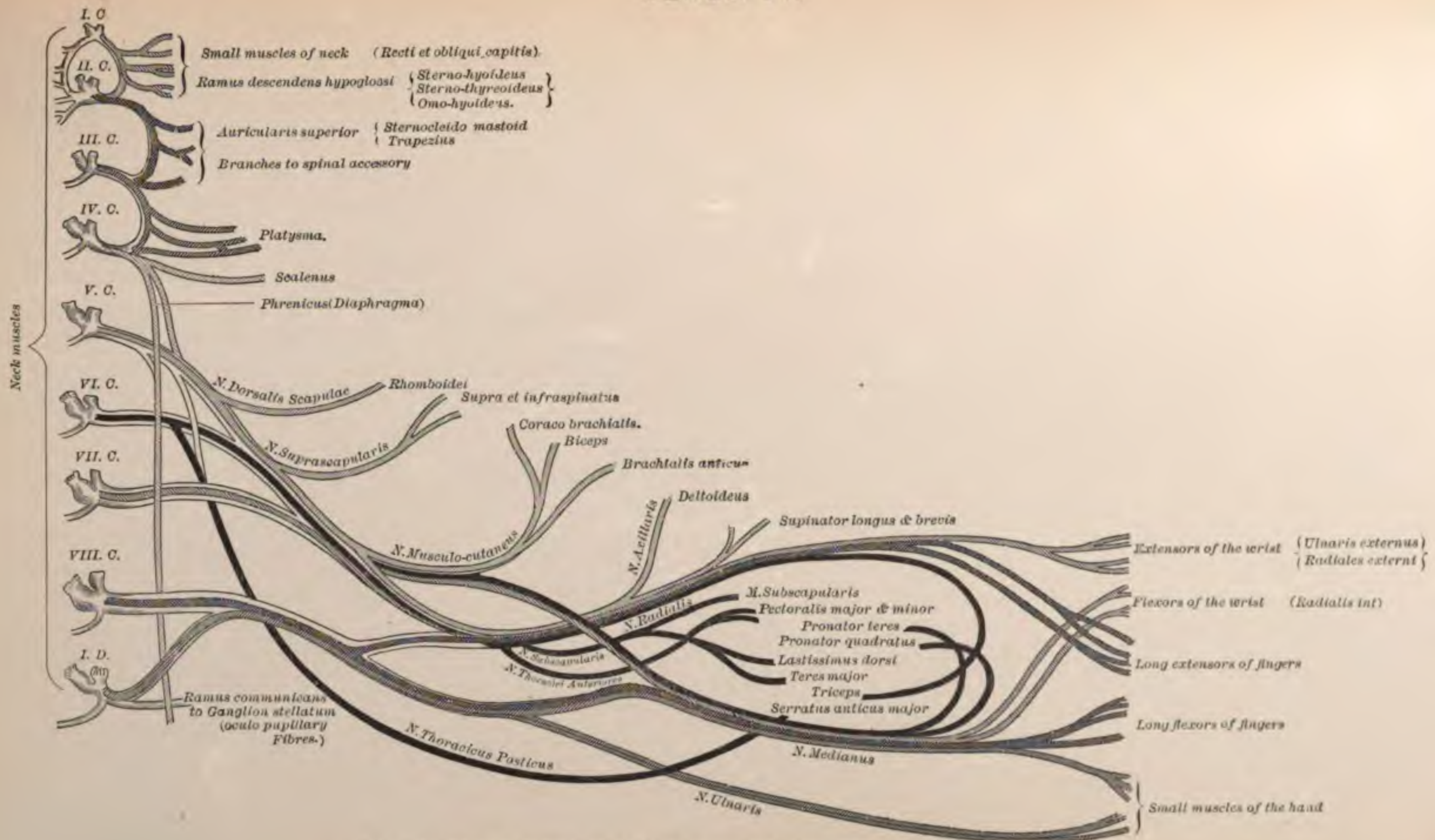
The paralysis following a nerve injury or neuritis requires electrical treatment. In the majority of such cases the faradic contractility in the muscle is lost and it is useless to apply faradism. The galvanic contractility, however, is always preserved, even though it may be diminished. For, by a strong galvanic current, even in the worst cases, a fair contraction can be produced in the paralyzed muscle when the current applied to the muscle is interrupted by means of the interrupting electrode held in the hand. The pole to be used over the muscle should be that to which the muscle responds most quickly. If a complete reaction of degeneration is present the positive closure contractility will be greater, and then the positive pole should be placed upon the muscle. If a partial reaction of degeneration is present the negative closure contractility will be greater, and then the negative pole should be used. The other pole is to be applied to the limb higher up in the course of the nerve. The application should be made daily and should last about five minutes to each muscle. The object of such applications is to exercise the paralyzed muscle. It is not probable that the effect of the electricity is in any way to increase the repair of the nerve or to aid its regeneration, hence there is no object in giving a continuous current to the nerve, as some authors advise. The electrodes should be of sponge and covered with clean gauze, and wet with warm salt water.

The paralysis may be also aided by massage and careful manipulation of the joint which the paralyzed muscles should move. In cases of spontaneous neuritis where the nerve is very tender, massage may be so painful as to be injurious, and it should always be remembered that massage, to be useful, should be painless. I have seen severe injuries of nerves follow the painful manipulation of unintelligent masseurs and osteopaths, and the statement which such individuals often make, that if their manipulations cause pain they are thereby doing good, is absolutely false. The object of massage in paralysis is to increase the nutrition and circulation in the paralyzed muscles. It should be done gently for about one-half hour daily, and should be followed by a sense of warmth and comfort, and not by fatigue.

A secondary result of the paralysis of certain muscles is the contracture of their healthy opponents, producing forced positions in a

The Gardner Sanitarium

PLATE IV.



The Cervico-brachial Plexus and its Branches. (Kocher.)

joint and very often quite serious deformities. The joint may even become stiff or the bones may become displaced. Thus in wrist-drop which follows musculospiral palsy the intrinsic bones of the hand are not uncommonly displaced, so that a distinct protuberance upon the back of the hand becomes visible. Manipulations of such displaced or stiffened joints are very valuable, and should be urged from the very beginning, in order to prevent deformity or stiffness.

The trophic symptoms which occur in neuritis usually yield at once to a careful disinfection of the skin and protection of the skin from extraneous injury or infection. This can be attained by washing the parts in a solution of bichloride or of carbolic acid, applying carbolized vaseline to the surface, and wrapping in absorbent cotton. It is very important to prevent the occurrence of these trophic symptoms by proper care of the skin from the very beginning, and patients suffering from neuritis or having any anæsthetic regions should be warned against the possibility of unnoticed injuries or burns.

INJURIES OF SPECIAL NERVES AND SPECIAL FORMS OF NEURITIS.

The affections of the cranial nerves will be considered in connection with diseases of the brain.

There are thirty-one pairs of spinal nerves which are named after the segments of the spinal cord from which they arise, there being eight cervical, twelve dorsal, five lumbar, five sacral, and one coccygeal. The origin, course, and branching of these nerves are shown in Plates IV. and VI. It is not needful to describe the anatomy of these nerves, but it is necessary to consider certain special types of neuritis and the results of injuries to certain nerves which are frequently met with in practice.

Cervico-brachial Neuritis. The anatomy of the cervico-brachial plexus is shown in Plate IV.

Etiology. A neuritis of the cervico-brachial plexus may occur in the adult as the result of injuries to the neck and shoulder, especially after dislocations, as the result of spinal caries, or of tumors, or aneurisms growing in the neck; as a sequel of any of the infectious diseases, especially grippe, or of toxic conditions; from rheumatic and gouty states, and also from taking cold. It is more frequently met with in women, and persons above the age of forty years are more liable.

Symptoms. If the neuritis is limited to the upper four cervical nerves a very intense occipital neuralgia is produced. The pain runs up the back of the neck and over the head as high as the vertex, and is attended by extreme sensitiveness of the great occipital nerve to pressure; by tingling and numbness, and by difficulty in the movement of the head because of the pain produced by motion. The head is often held stiffly on this account. Occasionally the hair of the scalp comes out. There is sometimes a weakness or paralysis of the deep cervical muscles.

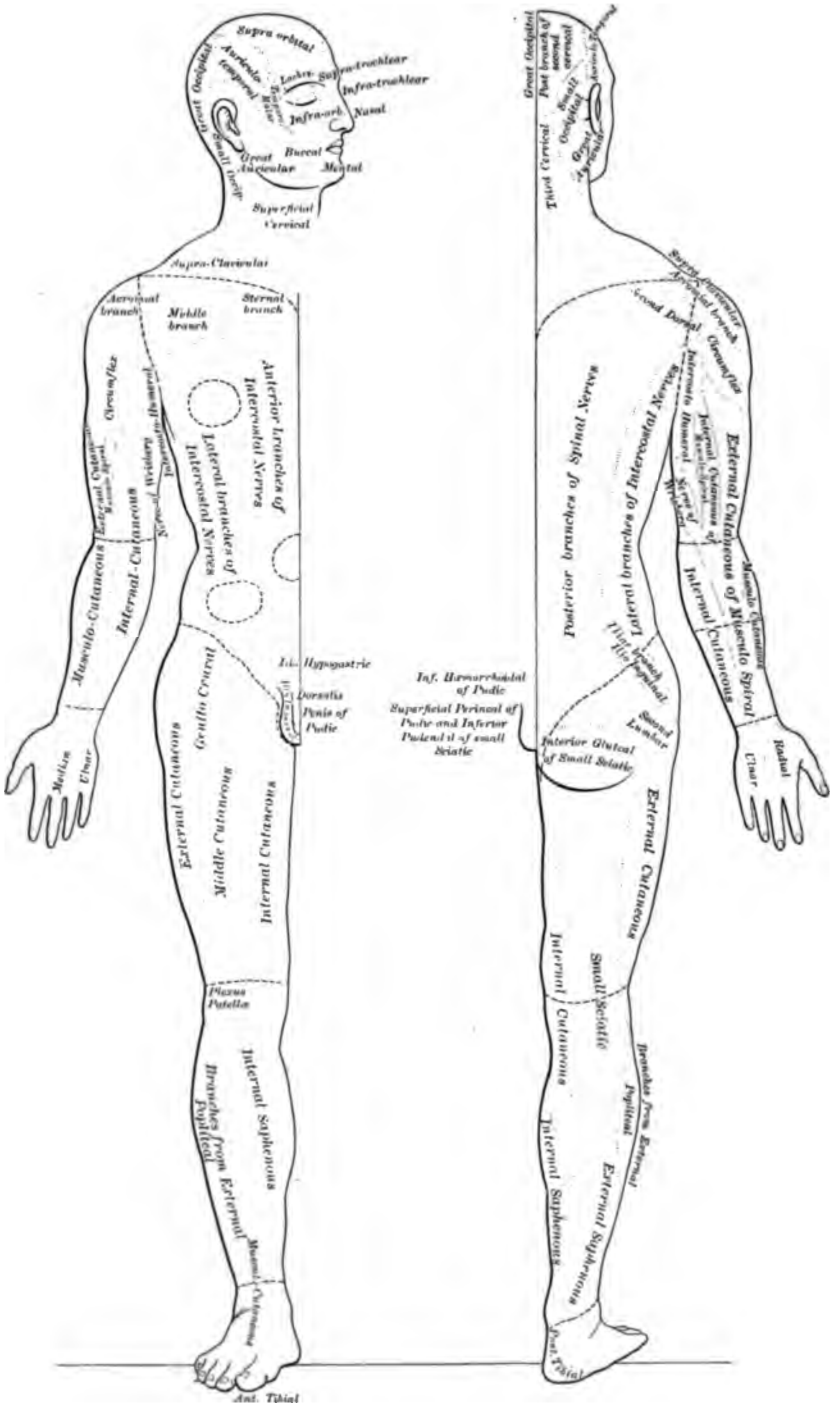
If the neuritis is limited to the fifth and sixth cervical nerves or their branches in the plexus, pain is very intense in the neck above the clavicle, and is felt over the shoulder and in the axilla and down the back of the arm, and even into the forearm, and the paralysis affects the deltoid, biceps, coracobrachialis, and supinator muscles.

If the lower cervical nerves and the first dorsal nerve are the seat of the neuritis the pain and tenderness are very intense over the clavicle, and the pain is felt down the front of the arm over the entire forearm and in the hand and fingers, and the paralysis affects the muscles of the forearm and hand.

It is to be remembered that the spinal origin of the sympathetic nerve in the neck is in the last cervical and first dorsal segments of the spinal cord, and the anterior nerve roots coming from these segments contain the fibres passing to the cervical sympathetic. These nerve fibres leave the main cervical nerves soon after their exit from the vertebral foramina and ascend, lying deeply beneath the muscles. In the majority of cases of neuritis of the lower part of the brachial plexus they are not involved, but occasionally, if the inflammation extends deeply through the spinal nerve roots, or if these nerve roots are compressed by tumor, or are involved in caries of the spine, or are lacerated within the spinal canal, the symptoms of paralysis of the sympathetic appear. These symptoms are a slight retraction of the eyeball and narrowing of the palpebral fissure, the eyelids appearing partly closed and the outer angle of the eye being slightly drooping. The pupil is somewhat contracted and does not dilate fully when the eye is shaded. There is a slight pallor of the side of the face and neck, an unusual dryness of the nostril and mouth on the side of the injury, and a diminution in the secretion of sweat on the neck, arm, and chest of the injured side. If the patient is subjected to heat the injured side does not flush and perspire in the normal manner.

The existence of these symptoms of injury of the sympathetic, to which attention was first directed by Klumpke, is sometimes of much importance in diagnosis, as an indication that an injury of the plexus is present. Thus in a patient seen with Hartley at the New York Hospital, who had fallen several stories and sustained a fracture of the right side of the cranium and a manifest strain of the left arm, it was important to determine whether the paralysis of the left arm was due to a laceration of the brachial plexus or to an injury of the brain. The existence of great pain on pressure over the brachial plexus and on motion of the arm, with almost total anæsthesia of the hand and arm below the elbow, and the presence of the symptoms indicative of injury of the sympathetic nerve in the neck, made it evident that the paralysis of the arm was due to a laceration of the nerves of the brachial plexus and not to a cerebral injury. The

PLATE V.



The Distribution of Sensory Nerves in the Skin.
(After Flower.)

The areas of the skin supplied by the cutaneous nerves are shown in finely dotted outline. The circles on the trunk show areas occasionally anæsthetic in hysteria. The lines across the limbs at ankle, knee and thigh, wrist, elbow and *shoulder show* the upper limits of anæsthesia in multiple neuritis of varying *degrees of severity.*

development of the reaction of degeneration in the muscles in the course of ten days confirmed this diagnosis. Usually in brachial neuritis several cords of the plexus are involved simultaneously, and then the pain radiates over all the branches of the nerves which arise below this plexus. Plate IV. shows the formation of this plexus from the nerve roots and the distribution of the cords of the plexus to the various nerves of the arm. Plate V. demonstrates the distribution of the cutaneous nerves in the skin of the arm.

A comparison of the symptoms developing in any case, namely, the distribution of the pain, of the lines of tenderness, of the areas of tingling, numbness, and anæsthesia, with these diagrams will serve to indicate what branches of the brachial plexus are involved in the neuritis. As cases differ widely from one another in the extent of implication of the nerves, it is only by such a comparison of each case with a diagram that a definite diagnosis can be reached.

Brachial neuritis of the ordinary form in the adult, due to exposure to cold, is an extremely common disease, fully as common as sciatica. It is an exceedingly painful affection, coming on sometimes with great suddenness, as in a case described by Klumpke[1] where the autopsy showed an extensive hemorrhage in the sheath of the nerves. Hence it has been called "apoplectic neuritis." Its onset may be gradual through several days, under which circumstances an extreme congestion with interstitial neuritis and secondary implications of the nerve fibres are present. A pain in the location of the plexus and pain radiating outward are the most distressing symptoms, and are so intense and agonizing as to deprive the patient not only of all use of the limb, but also of sleep. Soon after the onset of the pain any motion becomes so uncomfortable as to make rest of the limb imperative. The numbness and tingling which attend the pain are extremely disagreeable and are more intense after the disease is well established at the end of a week. The paralysis of the muscles supplied by the affected nerves develops within three or four days of the onset, and these muscles become atrophied as the case goes on, and usually after ten days show a reaction of degeneration. In brachial neuritis trophic disturbances of the skin, glossy skin, herpes, eruptions of various kinds, and interference with the growth of the nails are very common occurrences. The skin is manifestly thin, becomes very shiny and smooth, appears as if tightly stretched over the fingers, hand, or arm, and is exceedingly tender and hypersensitive to touch, to heat, and to cold. Sensations caused by moving the fine hair on the skin are peculiarly disagreeable. The limb is very frequently covered with perspiration, which may be of a fetid odor. Such a neuritis usually persists for several weeks or even months. The average duration in ten such cases under my observation was four months. The symptoms gradually subside, but the weakness often persists for some weeks after the pain has disappeared.

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[1] Rev. de Méd., 1885.

Pathology. Pathological observations in cases of neuritis of the brachial plexus are comparatively rare, as the patients usually recover. Klumpke's case of hemorrhage within the sheath of the plexus has already been mentioned. Jacobson reports a case following carcinoma of the breast of nine months' duration. A microscopic examination showed an almost total disappearance of the nerves of the brachial plexus, though there was no infiltration with carcinomatous masses. The process of degeneration was present in many bundles, with a manifest disappearance of nerve fibres, and an atrophic condition was found in the peripheral nerves of the arm. The cells of the posterior spinal ganglia in the cervical region showed degenerative changes by the Nissl method of staining, and by the Marchi method there was discovered a manifest degenerative change in the posterior root zone of the spinal cord corresponding to the entrance of the nerve roots from which the brachial plexus was made up. Degeneration ascended in the column of Burdach, and also descended in the comma-shaped bundle of Schultze as far down as the fifth dorsal segment. This observation proves that in some cases when the sensory neurone is involved in one of its branches the cell body and the other branch may also degenerate.

Prognosis. The prognosis is fairly good, as recovery eventually occurs in the majority of the cases. But the progress is slow and is attended by very great discomfort. It is to be remembered that nerves in regenerating grow but one centimetre a week, and hence the repair in the affected nerves requires a long time, especially if those nerves are the long ones to the fingers. In the traumatic cases the prognosis is less favorable than in injuries of single nerves. Thus in a case seen with McBurney, a calcareous cicatrix had involved the three cords of the plexus and destroyed the nerves so completely that nothing could be done to induce repair.

Treatment. Treatment consists of absolute rest, the arm being carried in a sling and held to the side by bandages, constant application of heat, either by packing the shoulder and arm with cotton, which is to be covered with oiled silk, hot bottles being applied outside of the bandage, or by the application of poultices. In my experience cold applications—ice packs, or spraying with ether, or chloride of ethyl, which are recommended by the Germans—produce great discomfort and are voluntarily discarded by the patients in favor of hot applications. The use of the actual cautery in producing counter-irritation over the painful nerve trunks is urged by all authorities, and I have certainly known it to alleviate the pain. It may be done daily if the touch made is very light, so as not to break the skin, merely producing a red line upon it. Electricity may be used, and in some patients appears to give considerable relief. A very mild galvanic current, from four to six milliampères only, should be applied with the positive pole over the painful region, and the negative pole over the back of the neck. The current should not be broken and should be allowed to pass for ten minutes through the painful region, the pole being shifted in order to bring all of the painful parts within the influence of the current. In applying the electricity to the arm, Remak recommends that a current be used diagonally through the painful nerve, the positive pole being over the most painful point and the negative upon the other side of the limb. During the early and painful stage of the disease any electrical application producing a contraction of the muscles is too painful to be endured, but later in the course of the case, when the pain is subsiding, it is well to exercise the muscles by electricity in order to keep up their tone until the nerve has regenerated. The application of electricity has no effect upon the course of the case or upon the regeneration of the nerve trunk.

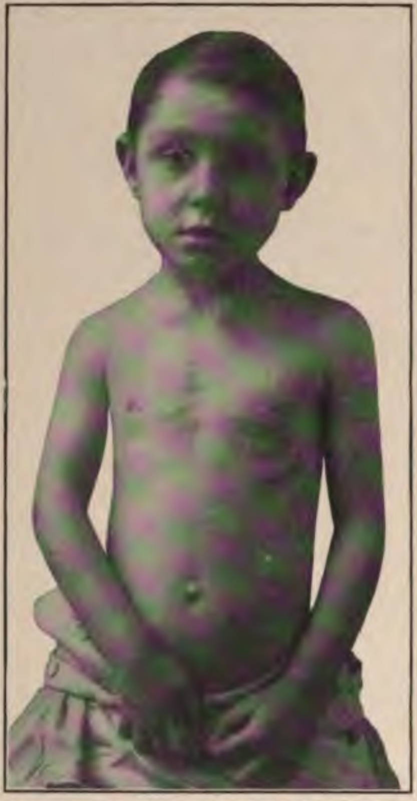
Massage of the limb is advisable when the parts are not too tender to prevent its use, and as soon as the tenderness subsides sufficiently to allow of it, it should be begun and continued until recovery. Some relief is often obtained by douching the limb with hot water from a spray or by alternate douches of hot and cold water. Patients differ in their susceptibility to cold in this condition. Many patients are hypersensitive to cold in conditions of neuritis, and under these circumstances cold water should be avoided.

The pain in brachial neuritis is so intense as to require medical relief. The various analgesic preparations—acetanilid, antipyrine, phenacetin, alone or combined—should be tried before codeine or opium are resorted to. But in the majority of cases in this condition it is necessary to use hypodermic injections of morphine, and these may be used very freely in sufficient amount and with sufficient frequency to secure relief from the intense agony which otherwise exhausts the patient. Care should be taken to keep up the general nutrition of the patient during the course of the disease. Good food, adjuvants to digestion and laxatives which counteract the action of the morphine should be used freely, and it is well to insist upon the use of fatty foods in excess, as these appear to have a beneficial effect in increasing the rate of regeneration in the nerves. Alcoholic drinks should be avoided or used in great moderation. Strychnine and arsenic are of benefit in the stage of recovery, but are not to be used while the pain is intense. The glycero-phosphates of lime and soda in 15-grain doses, three times a day, are of much use.

Surgical treatment is demanded in traumatic cases which show no tendency to recover after three months. The plexus should be exposed, cicatricial tissue removed, and a union of the separated nerves secured by a nerve flap, or by inserting the ends in a tube of decalcified bone.

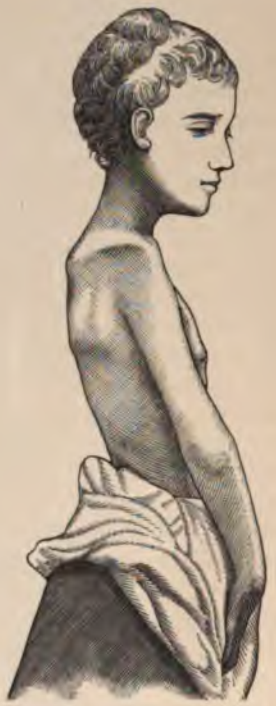
Obstetrical Paralysis, or Birth Palsy of Duchenne. This form of paralysis is noticed within a few hours or days of the birth of an infant. It is usually limited to the deltoid, biceps, brachialis anticus, supraspinatus and infraspinatus, and supinator longus muscles, but occasionally involves the extensor muscles of the wrist and fingers. The arm hangs loose at the side of the body, its position as a whole being governed by gravitation, with the forearm extended and pronated, and the wrist and fingers flexed. (See Fig. 14.) If the arm be lifted and then let go, it falls into this position. The muscles which are paralyzed are relaxed and their opponents are not rigid, so that all joints are freely movable and motion does not give the child pain. The only movements in the paralyzed arm are those of extension of the forearm and motions of the wrist and fingers. Sometimes when the extensors of the wrist are involved there is slight extension of the two distal phalanges only. If the paralyzed muscles are examined electrically the reaction of degeneration can be demonstrated, but this is often difficult in an infant on account of the undeveloped condition of the muscles and the thick layers of fat. A very strong current is necessary to produce the reaction, and this is, of course, attended by pain; hence electrical

FIG. 14.



Boy, aged seven, whose right arm had been paralyzed since birth. The position of the arm and hand and the atrophy are typical.

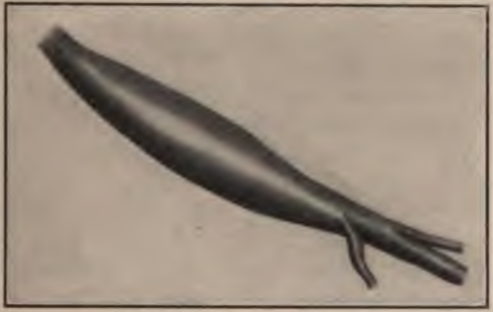
FIG. 15.



Extreme atrophy of right shoulder and arm due to birth palsy. (Dejerine.)

examinations are difficult in infants. If such tests are made the normal arm should be taken as a standard for comparison. If the sensibility is tested by a needle it will usually be found to be considerably impaired over the area of the arm corresponding to the cutaneous branches of the circumflex nerve—a heart-shaped area whose point corresponds to the insertion of the deltoid muscle, and sometimes, also, along the outer side of the arm and forearm in the distribution of the musculocutaneous nerve. After this condition has remained for several weeks or months without much change, as it often does, the muscles which are paralyzed become considerably atrophied and feel unduly soft and yielding. (Fig. 15.) The actual size of the limb, however, may not be very perceptibly reduced, for the fat in a thoroughly healthy baby is much greater than the substance of muscular tissue, and, therefore, there is not the rapid wasting of the arm and forearm which is noticed in adults suffering from paralysis of the brachial plexus. When the condition has lasted three or four months there sometimes appears a slight stiffness of the unparalyzed muscles, so that bending the forearm and wrist or opening the little closed hand is not as easy as at the beginning. A permanent rigidity, however, rarely if ever develops. Many of these cases go on to spontaneous recovery, which ensues within six months after birth. Others remain longer and do not recover within the first year. The condition may remain for three or four years and pass away only when the child is old enough to be taught systematic gymnastic exercises. I have seen one case where no recovery had ensued after seven years, and then an operation by Dowd demonstrated the existence of a cicatricial mass at the junction of the roots of the fifth and sixth cervical nerves in the neck, which had prevented regeneration. (Fig. 16.) This was too extensive to warrant removal. I have seen one girl of fourteen who still had complete disability of movement at the shoulder-joint and inability to supinate the arm, due to the occurrence of birth palsy.

FIG. 16.



Thickened cord of brachial plexus at the junction of the 5th and 6th cervical nerves found at operation on boy shown in Fig. 14. (Natural size, from a sketch by Dowd.)

The course of the disease and the final condition depend upon the severity of the lesion.

The lesion in all cases of birth palsy involves the upper two nerves of the brachial plexus. Duchenne showed that pressure backward upon the side of the neck is liable to compress these two nerves against the lamina of the sixth cervical vertebra. Erb has shown that it is possible by a careful examination to find a spot two centimetres above the clavicle, back of the outer edge of the sternomastoid muscle, corresponding to the point of emergence of the sixth cervical nerve between the scaleni, at which point irritation by the faradic current will produce a contraction in the deltoid, biceps, brachialis anticus, and supinator longus muscles; and if the irritation be increased the extensors of the wrist will also contract. Pressure upon this particular region is often made during delivery, either by the clavicle or by forceps or by the fingers of the obstetrician. This is more common when there is a breech presentation and the after-coming head is extracted in the common method. The index and middle fingers of the left hand being open like a fork over the shoulders of the child, traction is commonly made upon the shoulders, and the pressure of the obstetrician's finger in the neck often produces injury of the plexus. In some cases injury of the plexus is produced by attempts to bring down the hand or arm in breech presentations or to replace these when the head presents. Forceps applications in an awkward position may also produce this injury. Children do not appear to be very liable to injury of the nerves in spite of the many accidents which befall them. I have not seen a case of traumatic neuritis in the plexus of a child below the age of fourteen years, excepting the form of brachial neuritis just described. I have seen this form in adults due to pressure, in one case in a piano-carrier, and in another in a laborer.

Treatment. The arm should be kept in a sling, with the elbow flexed, and should not be allowed to hang down, since its weight often suffices to overstretch the ligaments of the shoulder which are no longer assisted by the action of the deltoid. The entire arm should be rubbed daily, the mother being taught the kneading process of massage. It should be bathed freely night and morning with hot and cold water, so as to increase the general circulation and nutrition. Unintelligent people will be more likely to keep up the necessary rubbing if some simple lotion or ointment be ordered. As soon as any voluntary motion can be made the child should be encouraged to make it, even if it be necessary to bind the unaffected limb to the side of the body. As the child grows older systematic exercises of a gymnastic kind should be insisted upon daily. Galvanic treatment in accordance with the regular methods described on page 56, should be begun early and used until recovery has occurred. The mildest current which will produce contraction in the muscles should be used, that the treatment may not be too painful, and an intelligent nurse or mother can be instructed to carry out this treatment at home.

Paralysis of the Circumflex Nerve. Injuries of the shoulder and dislocations of the shoulder sometimes produce a paralysis of the circumflex nerve. The symptoms consist of pain and tenderness in the course of the nerve; paralysis and atrophy of the deltoid muscle, which prevents abduction of the arm, a relaxation of the ligaments of the shoulder-joint, as the deltoid muscle keeps the humerus in apposition to the socket; and an area of anæsthesia, triangular in shape, with the apex of the triangle downward corresponding to the insertion of the deltoid. (See Plate V.)

A patient under my care had been in the habit of standing in his office and resting his shoulder against the sharp edge of a mantel. About the first of May he began to suffer from pain in the back of the neck and about the shoulder, and after ten days his deltoid became suddenly paralyzed. This paralysis remained, in spite of treatment by electricity and massage, until the first of November, though by the middle of July he had recovered power enough to raise the arm above the head. The anæsthesia over the shoulder disappeared within the first month.

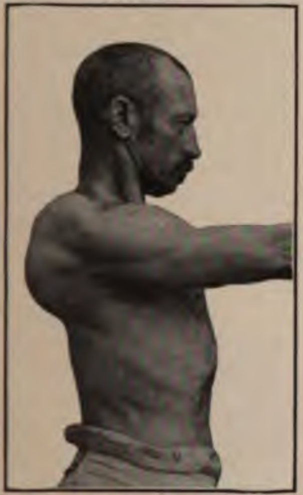
Paralysis of the Suprascapular Nerve occasionally occurs after dislocation of the humerus, or may be produced by falls upon the shoulder or on the hand, causing contusion of the shoulder. The symptoms are chiefly referable to paralysis of the supraspinatus and infraspinatus muscles. I have seen this following removal of deep cervical glands in the neck by accidental division of the nerve trunk. The shoulder is very much hampered in its movement, as is also the arm. When the arm is abducted and raised the head of the humerus falls from lack of support by the supraspinatus muscle; hence the arm cannot be held up. Movements of outward rotation are also impaired by the paralysis of the infraspinatus. The scapula is slightly rotated, its lower angle being moved upward and inward, and its upper angle projecting on the side of the neck. The defective action is somewhat supplemented by the deltoid and the teres minor, which muscles occasionally become a little hypertrophied in the course of the case. Sometimes a small region of anæsthesia is found over the scapula.

Neuritis of the Shoulder-joint. **Painful Shoulder.** A condition of extreme pain and stiffness at the shoulder-joint, due to a neuritis of the nerves within the joint, is not an uncommon affection. It occurs, as a rule, in women of middle age, but men are not exempt. It occurs in gouty and in anæmic and cachectic individuals, and is often seen in old age. It sometimes appears to follow an exposure to cold, and sometimes it can be traced to a slight injury. It begins rather suddenly with pain in the shoulder-joint, but without any swelling or exudation within the joint and without the appearances of rheumatism or of arthritis. Very soon any movement becomes extremely painful, and as a result the muscles about the joint are thrown into a state of tonic spasm. The arm is adducted tightly to the chest, and while slight forward or backward motions are possible, any attempt at abduction is followed by an immediate contraction in the pectorals and latissimus dorsi. There are tender points in front and at the back of the joint, and pressure of the head of the humerus into its socket is very painful. The condition remains for many weeks, and as the result of the immobility the deltoid frequently becomes atrophied. Occasionally I have seen an anæsthesia in the circumflex area of the skin and such a rapid atrophy of the deltoid as to suggest a neuritis of the circumflex nerve, but this is not an invariable accompaniment of the affection. When there are no objective signs of neuritis in the shoulder, or of tenderness about the nerves in the neck, the affection may be thought to be hysterical, as neurotic individuals appear to be more subject to it than others. But the lack of remissions in the disease and the fact that the spasm of the muscles does not appear to relax under chloroform—a fact that I observed in a patient who had an operation for cancer of the breast while this affection was in progress—leads me to believe that the spasm is a reflex one from the nerves within the joint and that the origin of the trouble is a neuritis of these nerves. As any motion is extremely painful, patients guard the arm by carrying it in a sling and by bandaging it to the chest. They find hot applications and packing in cotton agreeable, and they usually obtain considerable relief from the application of a Paquelin cautery to the front and back of the shoulder. It seems to have less effect when applied over the region of the deltoid muscle. The treatment is rest, cautery, and the use of sedatives, including codeine, if necessary, for the pain. When the acute symptoms are over, massage of the muscles may be attempted, but movement of the joint is to be avoided, as it is liable to produce a return of the spasm in the muscles. In all cases that I have seen, recovery has eventually occurred, but from four to six months have elapsed before the full use of the arm has been restored.

Paralysis of the Posterior Thoracic Nerve. Injuries to the side of the neck sometimes bruise the long posterior thoracic nerve which passes to the serratus magnus muscle. I have seen two cases of this kind occurring in porters who had carried a piano upon the shoulder; and several cases from injuries to the side of the neck. I have seen a case resulting from the effort of swinging a hammer. Falls and blows upon the neck or shoulder which cause extreme extension of the head may also produce this. And many cases have been noticed after exposure to cold.

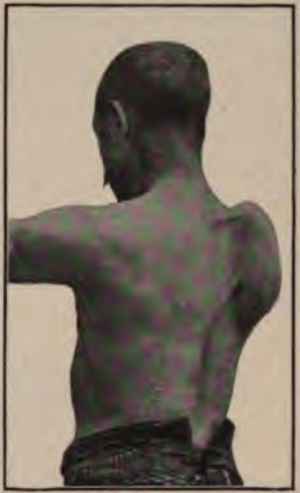
The symptoms of the disease are severe pains about the shoulder and neck and total paralysis of the serratus magnus muscle. It will be remembered that the function of this muscle is to hold the posterior edge of the shoulder-blade against the chest and to rotate the shoulder-blade in the act of raising the arm. When it is paralyzed the shoulder-blade projects from the chest like a wing in a very characteristic manner (Figs. 17, 18, 19), and the arm cannot be lifted much above the horizontal level when the forearm is extended. Motions of the forearm forward and backward are also somewhat impaired. If the arms be abducted, forcible inspiration shows a defective expansion of the chest on the paralyzed side. An electrical

FIG. 17.



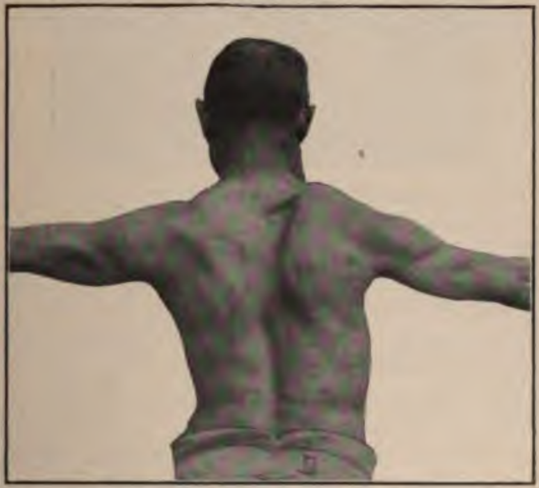
Paralysis of the serratus magnus muscle, causing displacement of the scapula when the arm is held forward. (Icon. de la Salpêtrière.)

FIG. 18.



Paralysis of the serratus magnus muscle, causing displacement of the scapula when the arm is held forward.

FIG. 19.

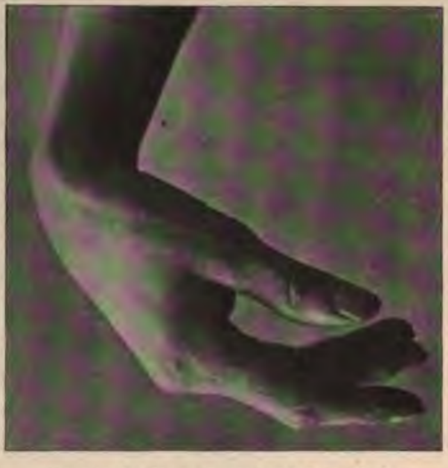


Paralysis of the serratus magnus from injury of the posterior thoracic nerve. Position of the scapula when the arm is abducted. Further elevation of the arm is impossible, as rotation of the scapula is impossible.

examination usually demonstrates a reaction of degeneration in the serratus magnus muscle. The motor points of this muscle are on the side of the chest beneath the axilla. A slow recovery usually ensues, provided the nerve is not entangled in the scar at the point of injury. If recovery fails to occur after several months, a surgical exploration is justifiable.

Paralysis of the Musculospiral Nerve. This nerve is injured more often than any other of the nerves of the arm, inasmuch as it is exposed in its passage around the humerus just below the insertion of the deltoid. Pressure of the head lying upon the arm, which frequently occurs in the sleep of intoxication, is sufficient to cause musculospiral palsy, or the compression of nerve in the axilla, if during sleep the arm hangs over a sharp edge of a bench or bed. The pressure of a crutch may also produce this form of paralysis. In Germany this is called "police paralysis," because of the method used in securing the arms of a prisoner by placing a bar across his back and beneath his two elbows and tying the forearms firmly to the arms. Gowers describes three cases which have followed violent contraction of the triceps—one in pulling on a pair of tight boots; one in throwing a stone, and one in grasping a support to prevent falling. I have seen cases which have developed from pressure during a long-continued anæsthesia, the patient coming out of the anæsthetic to find his arm paralyzed. I have also seen the nerve injured in fractures of the humerus. In one patient the administration of a hypodermic injection in this region was followed by paralysis.

FIG. 20.



Drop-wrist due to musculospiral paralysis.

The symptoms produced by musculospiral palsy are drop-wrist, with pronation, due to paralysis of the long extensors of the wrist and long extensors of the fingers and thumb, and weakness in flexion of the elbow from paralysis of the supinators. If the nerve is injured in the axilla the triceps is also paralyzed. Sometimes in very slight cases the supinator longus is not paralyzed. The position of the hand in drop-wrist is shown in Fig. 20. The action of the flexors of the hand is apparently weak in grasping objects, but if the wrist be extended by the examiner and held firmly in this position, the flexors are found to act in a normal manner. The awkward action of the forearm gives the patient considerable discomfort, especially the loss of power of supination. After the drop-wrist has continued for some days a prominence appears upon the back of the hand as a result of the overflexion of the wrist, due to a partial dislocation of one of the bones of the hand. But this subsides when recovery has taken place. The muscles paralyzed show the reaction of degeneration, excepting in very light cases. In the early stage of the affection some tingling and numbness may be felt over the back of the hand at the base of the index finger and thumb, and an impairment of sensibility may often be demonstrated in this region. (See Plate V.) This, however, rapidly subsides. Musculospiral paralysis can hardly be mistaken for any other disease, though drop-wrist may develop in any form of multiple neuritis, especially in lead palsy. It is then, however, bilateral, and the supinator longus usually escapes.

The prognosis is good for recovery, but this usually requires from three to five months.

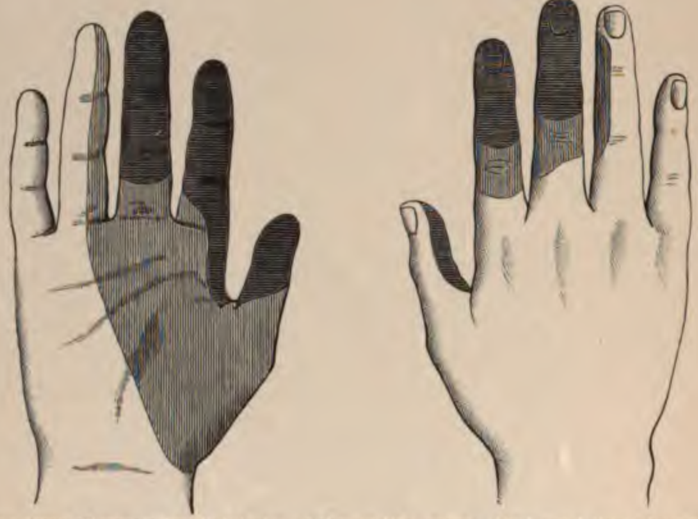
The treatment is that for ordinary traumatic neuritis.

Paralysis of the Median Nerve. This may occur from wounds or injuries of the forearm or of the brachial plexus, also from pressure of a crutch. The median nerve supplies the pronators, the flexor carpi radialis, the flexors of the fingers, and the muscles that abduct and flex and oppose the thumb and the two radial lumbricales, which flex the first phalanx; hence this injury produces paralysis in these muscles, resulting in a supination of the forearm and inability to flex the fingers firmly or to use the hand. The thumb cannot be abducted or opposed to the tips of the fingers. The anæsthesia produced is shown in Figs. 21 and 22. Pain is sometimes felt in the hand. When the nerve is injured at the wrist the action of the long flexors is not interfered with, hence the paralysis is limited to the fingers alone. There is usually some atrophy of the thenar muscles. Trophic changes on the skin and nails often occur.

The prognosis and treatment are in accordance with ordinary traumatic neuritis.

Paralysis of the Ulnar Nerve. The ulnar nerve is very frequently injured, as its course is an exposed one at the elbow and in the forearm. It may be affected by pressure in the axilla by a crutch, but this is rare. Exposure to wet and cold often causes ulnar neuritis. Long-continued pressure in a condition of anæsthesia, or even in the heavy sleep of intoxication, may cause paralysis of the muscles supplied by the ulnar nerve, and after dislocation and fractures of the arm or forearm it has been frequently observed. Callus, after fractures about the elbow, occasionally presses on and involves the nerve. The ulnar nerve supplies the flexor carpi ulnaris, the ulnar half of the deep flexors of the fingers, the muscles of the little finger, the interossei, the inner two lumbricales, and the adductors of the thumb.

FIG. 21. FIG. 22.

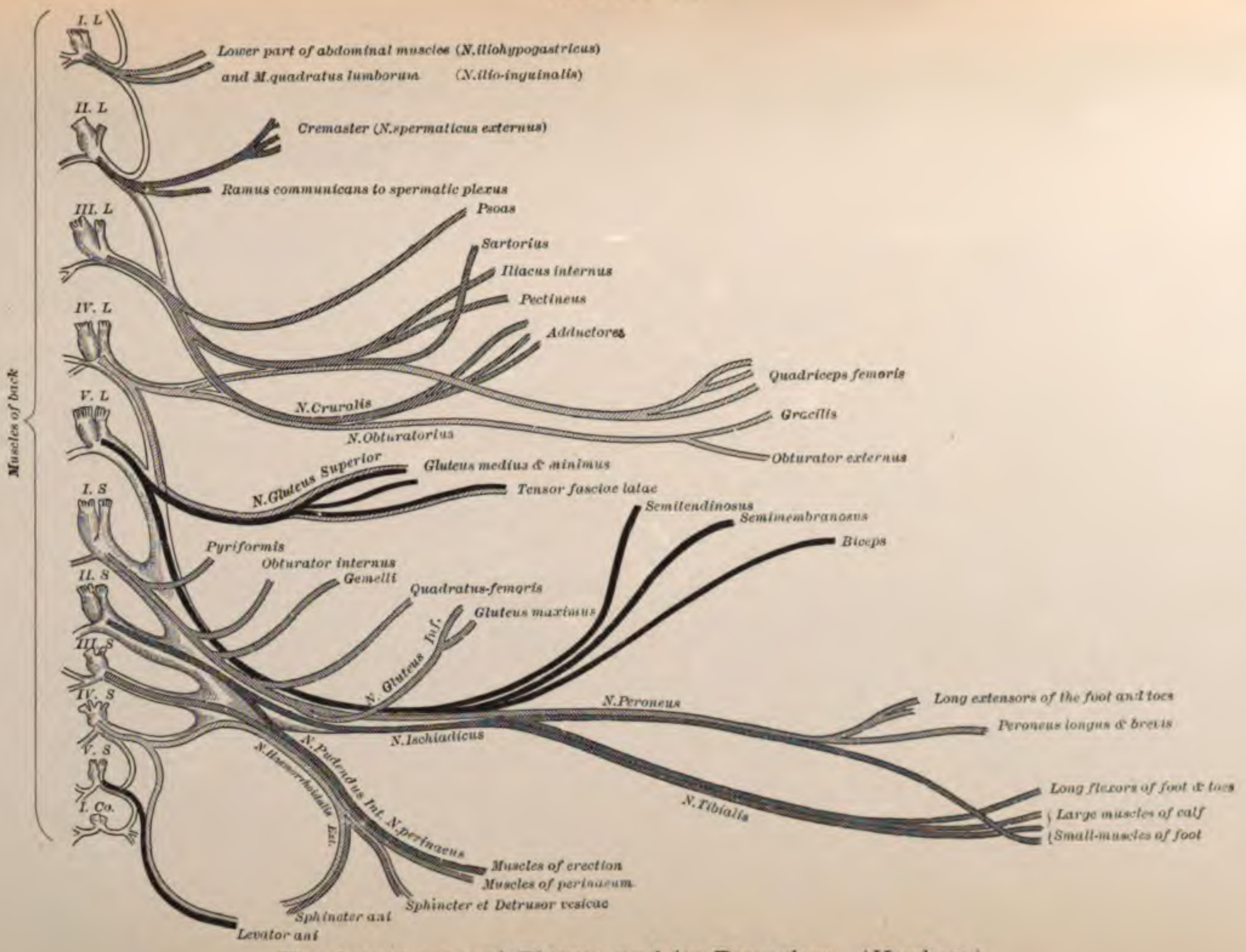


Showing areas of sensory loss in injuries of the median nerve. Horizontal lines show total anæsthesia. Vertical lines show partial anæsthesia. (Bowlby.)

The result of paralysis of the ulnar flexor of the wrist is not very manifest, as other muscles are capable of doing the work of the flexor carpi ulnaris. A paralysis of the flexor profundus digitorum makes the patient incapable of flexing the first phalanges of the little and ring fingers, hence there is no opposition to their extreme extension, and part of the deformity of the hand resulting from ulnar nerve palsy (Figs. 23 and 24) is due to this cause.

The chief disability, however, produced by ulnar nerve lesions is the paralysis of all the intrinsic muscles of the hand excepting the first and second lumbricales, which are supplied by the median nerve. As a result of this paralysis the use of the thumb and fingers is very much impaired, the thenar and hypothenar eminences become flat and flabby, the interossei of the hand are also atrophied, so that the bones and long tendons of the fingers stand out beneath the skin; and the little and ring fingers are flexed in their second and third phalanges because of the inability to extend them, due to the complete paralysis of the interossei. This is the second cause of the deformity produced in the hand. The extension of the middle and end phalanges of the other two fingers is also considerably impaired. The adduction and abduction of the fingers is completely abolished

PLATE VI.



The Lumbosacral Plexus and its Branches. (Kocher.)

and the thumb cannot be brought forward because of overextension of its first phalanx by the extensor longus pollicis.

The sensory disturbances produced by ulnar palsy are tingling and numbness and possibly pain in the inner half of the hand, in the little finger and one-half of the ring finger, and these paræsthesia are usually attended by anæsthesia, which may be total in the little finger, but is only partial in the hand and ring fingers.

FIG. 23.

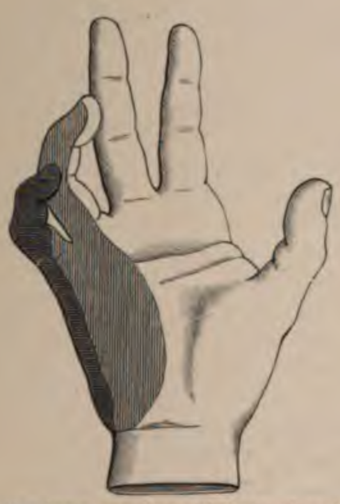
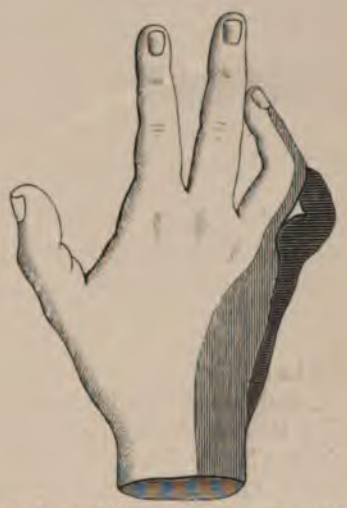


FIG. 24.



Showing sensory loss and abnormal position after injuries of the ulnar nerve. (Bowlby.)

The sensory disturbance is not always a total one, sensations of temperature and pain being very often retained when the sensation of touch is lost. Trophic disturbances are very liable to occur in the anæsthetic region of the skin.

The prognosis and treatment are those of general neuritis.

Neuritis of the Lumbar Nerves. The anatomy of the lumbar and sacral plexus is shown in Plate VI.

The lumbar plexus supplies the flexors and abductors of the thigh, the extensors of the knee and the cremaster muscle. Its sensory distribution is shown in Plate V.

The lumbar plexus rarely suffers from neuritis, as it is well protected from cold or from external injury. But tumors within the spinal canal, tumors in the abdomen, cancer of the spine, and psoas abscesses occasionally set up a neuritis or cause pressure on the nerves of the plexus. I have seen several cases following operations for appendicitis where filaments of the nerves were divided.

The symptoms may be limited to one or two muscles, such as those supplied by the obturator nerve, or the anterior crural nerve, or may be widespread. In the first case the legs cannot be crossed, as the abductors of the thigh are paralyzed and rotation of the leg is impaired. In the second case the extensors of the knee are weak and atrophied and the knee-jerk is lost. In both cases the inability is marked in walking, and the patients move with difficulty. There is usually pain in the groin and thigh, and sometimes this shoots downward into the genital region. There may be tenderness along the course of the anterior crural nerve. The degree of anæsthesia in the skin is slight, but tingling and numbness are felt in the sensory distribution of the plexus.

Paralysis of the External Cutaneous Nerve. The external cutaneous nerve of the thigh supplies the skin over the outer portion of the thigh, from the line of the trochanter downward to the knee. Occasionally this nerve is diseased and then, as a rule, there is a sensation of pain in its distribution, which is attended by paræsthesia and by diminution of sensibility in the skin. The affection runs the ordinary course of a neuritis, and should be treated as a peripheral neuritis. Attention was first called to it by Bernhardt, and hence it has been termed "Bernhardt's Paralysis."

Neuritis of the Sacral Plexus is a rare affection and is usually due to injury of the sacral plexus during a difficult labor. Every obstetrician has observed a number of cases in which during a delayed or difficult labor, the woman has been seized with sudden severe pains in one leg, usually in the back of the leg, and often below the knee, and has suffered from a flexor spasm of the leg for some hours during labor. A day or two after delivery paralysis of the muscles of the leg below the knee develops, and also a condition of anæsthesia extending down the back of the thigh and around the outer side of the leg and into the foot. In some cases the bruising of the nerve is a slight one and all the symptoms subside within three or four weeks. In other cases the injury is more severe, and months elapse before the paralysis subsides and the patient is able to walk. It is to be remembered that conditions of paralysis may follow labor from anæmic states of the spinal cord or from an infectious multiple neuritis. These conditions, however, produce paralysis in both legs, while the disease under discussion is always unilateral. In sacral plexus paralysis the functions of the bladder and rectum are usually normal, and the symptoms of neuritis, pain, tenderness, paralysis, reaction of degeneration, are limited to the distribution of the sciatic nerve. Tumors in the pelvis and caries of the sacrum produce the same condition.

Neuritis of the Sciatic Nerve. Sciatica, or neuralgia of the sciatic nerve, is considered on page 93, but there are many cases of so-called neuralgia which are actually due to neuritis, and there are cases of neuritis of the sciatic nerve which develop without any preceding neuralgia. The disease may be produced by any one of the many causes of neuritis and neuralgia, but seems to be more common as a sequel of the grippe than of any other infectious disease, and it also occurs with great frequency in gouty and anæmic individuals.

Symptoms. The symptoms are pain in the entire domain of the sciatic nerve and tenderness along the course of the nerve, which is not to be confounded with the existence of tender spots at Valleix's points present in sciatica. The nerve is found to be tender in its entire length and not infrequently localized swellings can be detected by palpation. The pain is usually constant as contrasted with the paroxysmal attacks of neuralgia. The patient is never able to get into an easy position so as to be free from pain. There is great restlessness and uneasiness in the entire limb, and all movement is extremely distressing. Walking can only be done with difficulty and pain and has to be avoided. Very often an actual paralysis develops in the muscles supplied by the sciatic nerve, namely, those below the knee, and an atrophy with reaction of degeneration may develop. Not infrequently the pain of the neuritis gives rise to reflex spasms in the muscles, and such twitchings of the muscles of the leg are liable to be exceedingly distressing and painful. The leg is frequently hot and the secretion of perspiration may be increased. I have occasionally seen glossy skin upon the outside of the leg and upon the foot and irregular growth of the toe-nails. The disease is very slow in its progress, and often remains for months before recovery follows.

Treatment consists of hot applications to the leg, which must be kept at rest, the patient being confined to the bed and the position of the limb changed frequently by the aid of numerous small pillows. It is a mistake, in my opinion, to apply a long splint to the limb, as has been recommended by Hammond, inasmuch as the strained position produces very great disturbance and discomfort. The tenderness of the limb secures a sufficient amount of rest to the part without this enforced bandaging. Hot applications may be kept up by poultices or by the use of hot bottles, but great caution is to be used in applying the latter, to avoid blisters or serious burns, inasmuch as the skin may be insensitive below the knee. Some patients experience relief from the use of electricity, the continuous galvanic current being employed, with the positive pole over the painful nerve, the negative pole being placed upon the back. The best method of treatment, however, is by means of the actual cautery, which often gives relief from the pain for several hours, so that the patients are glad to have the application repeated, even though it is painful. The various forms of hydrotherapy, especially a hot douche in a bath, are of service. As in the treatment of brachial neuritis, sedatives may be used freely. Hypodermics of cocaine in the upper part of the nerve are often better than morphine. When the condition is not improved by such treatment, puncture of the nerve by a needle has been known to give relief, probably by allowing fluid to escape from the sheath. Several needles may be passed into the nerve and allowed to remain for an hour. This must be done with great care so as to be aseptic. In chronic cases stretching the nerve has been done with some good results, but too much should not be promised, as the operation is uncertain. The sciatic may be stretched without an operation, the leg being extended and the thigh forcibly flexed on the trunk. This is usually a very painful procedure, requiring an anæsthetic. It is occasionally of benefit in those cases where an interstitial neuritis has caused adhesions between the nerve and its sheath.

Paralysis of the External Popliteal Nerve. The external popliteal branch of the sciatic nerve in its passage around the fibula is exposed to injury either directly from pressure, as by a tight garter, or from external wounds, or from dislocation or fracture of the fibula. The result of such injury is a paralysis of the tibialis anticus, of the long peronei muscles in the leg and of the extensors of the toes, causing a dropping and adduction of the foot and toes and lameness in walking. The entire leg is raised so that the toes do not trip the walker. Atrophy soon follows with reaction of degeneration. There may be a loss of sensation in the outer side of the leg and on the back of the foot. Prognosis and treatment are the same as in other forms of traumatic neuritis.

Paralysis of the Internal Popliteal Nerve is much more rare, as it is protected by its deep position under the knee. Occasionally it is strained or wounded. Then paralysis of the muscles of the calf of the leg and of the sole of the foot causes much difficulty in walking. The patient cannot rise on the toes or flex the ankle or toes. There may be pain, numbness, or anæsthesia in the outer part of the foot and in the sole.

Neuritis of the Plantar Nerves. The finer branches of the plantar nerves are not infrequently affected by neuritis. Such neuritis may be traumatic from walking in ill-fitting shoes, or upon very rough ground for an unusual length of time. It may be due also to taking cold, as when a person stands for some hours in the wet, or has the feet chilled and frozen. The symptoms are pain along the course of the nerves, between the long bones of the foot, great tenderness to pressure, and inability to endure the pressure of the shoe and inability to walk on account of the pain. Gowers pointed out the fact that propulsion of the body forward is done by pressure on the toes, and that when they are paralyzed the act of walking is seriously impeded. Not infrequently irregular areas of anæsthesia may be found in the sole, and the patient suffers from prickling and burning pain, as well as from sharp neuralgic pains. The sensory symptoms may be associated with motor symptoms of paralysis of the intrinsic muscles of the foot, so that the toes cannot be moved freely and the foot may become quite thin in consequence of the atrophy. Reaction of degeneration is not infrequently present. The affection must be treated by rest in bed, the patient not being allowed to walk about; by local applications of heat, and by the general measures recommended for the treatment of neuritis.

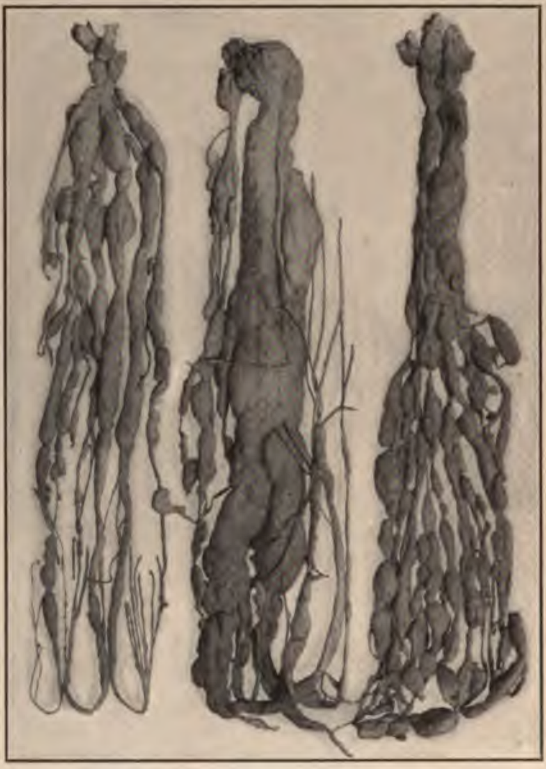
Morton's Toe. In some persons the second digital branch of the internal plantar nerve in its passage between the enlarged ends of the first and second metatarsal bones is compressed by anything which brings these two bones in close apposition. In others, any one of the digital nerves is similarly liable to be compressed. It seems probable that a congenitally abnormal position of the nerve is the predisposing cause of this affection. The disease develops in adults, and is usually the consequence of wearing shoes that are too narrow. The patient will be seized with a sudden sharp pain between the great and second toes, or between other toes, which is so agonizing in character as to prevent his taking another step, and which lasts until the shoe is removed and by a certain amount of manipulation the bones are separated and the pressure removed. But such attacks are liable to recur with great frequency, and to give rise to such distress and inability to walk as to require treatment. The disease is purely mechanical in its origin, and, therefore, anything which removes the pressure or which prevents the recurrence of the pressure will cure. In some cases it is sufficient to prescribe a very wide shoe. In others it is necessary to insert pads and strap them with adhesive plaster to the foot in such a position as to keep the metatarsal bones separated. Flatfoot may be the cause, and if corrected the pain may be obviated. But in some cases all these mechanical contrivances fail to give relief, and then resort to surgical treatment may be necessary. Morton was the first to operate for this condition, and hence it has been named after him. The operation may consist in a division of the nerve above the point of pressure, by an exsection of a portion of the nerve, but inasmuch as regeneration is sure to occur, this operation is unsatisfactory. The operation devised by Morton was exsection of the end of the metatarsal bone causing the pressure, and this has uniformly given relief.

TUMORS OF THE NERVES.

Neuroma. Any form of tumor—fibroma, angioma, sarcoma, etc., —may develop within a nerve sheath or upon it and thus produce an apparent tumor of the nerve. In such cases the nerve fibres may pass through the tumor or may be dissected apart by it and pass around it. Such tumors have been termed false neuromata, for they are not made up of nerve cells or fibres. But the fibres may become pressed by the new-growth and undergo degeneration. And the symptoms and course of the case under such conditions will be exactly such as have been already considered under compression of the nerves. Fig. 25 shows such false neuromata, which are very often multiple, as in this case.

Neuroma, or a tumor consisting of nerve fibres, first described by Virchow, is a rare form of tumor. The fibres of which it consists may be medullated or non-medullated; nerve cells are not found in such tumors; connective tissue exists in greater or less degree, so that some tumors are neuro-fibromata. There appears to be a tendency for such tumors to be multiple, and in the large majority of the recorded cases hundreds of minute tumors have developed. Occasionally the tumors have been large, but usually they are the size of a pea. When they appear on the sensory nerves of the skin they are easily felt and are usually tender. Under these circumstances they have been called "tubercula dolorosa." I have seen a patient with more than a hundred such tumors distributed over the entire body. Hoggan has shown that some tumors of this description were adenomata of the sweat glands. That some congenital tendency to the multiplication of nerve fibres is at the basis of this affection is proved by the fact that many of the cases reported have been in children. Thus in the so-called plexiform neuroma of the fifth nerve the disease is usually congenital and other nerves have been affected later in life. The tumors attain a certain size and then, as a rule, cease to grow; the condition remains permanent until death from some other cause.

FIG. 25.



Multiple neuromata. Nerves of the arm, the lumbar plexus, and the sciatic nerve. Specimen from the Museum of the College of Physicians and Surgeons.

In the majority of cases no symptoms are caused by neuromata, as the nerves on which the tumors develop are not affected by the growth. The tumors may be felt and may be tender to pressure, but are not subject to treatment and cannot safely be removed, as they are so numerous and as the nerves may be cut. Occasionally pain, hyperæsthesia, and numbness are caused by neuromata.

There is one form of neuroma which, however, requires special mention. It is the neuroma which develops on the cut end of a divided nerve, either after an amputation or after an injury without union. Such a neuroma may be extremely painful and tender and may cause pains referred to the peripheral termination of the nerve, and also spasms in the muscles related to these terminations. In such cases excision is imperative, but recurrence is not infrequent.

In one patient under my observation, who suffered from great pain referred to the toes, for many years after an amputation of the leg above the knee, excision of the neuroma on the sciatic failed to give relief. A portion of the sciatic nerve in the thigh was then excised, but this also failed to stop the pain. In this case it seemed probable that atrophic changes had occurred in the spinal cord, such as are known to follow amputations, and that these had involved not only the anterior horns, but also the sensory columns.

CHAPTER IV.

NEURALGIA.

Pathology. Etiology. Symptoms. Treatment. Special Forms of Neuralgia. Referred Pains.

NEURALGIA is a disease of a sensory nerve characterized by pain in the course of the nerve or in its peripheral distribution.

Pathology. As fatal cases are rare no autopsies are on record. But surgeons have cut out both nerves and ganglia for the relief of neuralgia, and from the examination of these much information has been obtained concerning the pathology of the disease. All the various processes of neuritis, with degeneration, and atrophy in the nerve fibres have been seen. But the more important lesions appear to be in the neurone bodies which lie in the ganglia. These neurone bodies have been found in different stages of degeneration, with chromatolysis, vacuolization, and atrophy. Some neurones stain only faintly and are most irregular in outline, being so shrunken as to leave the space in which they lie almost empty. Some neurones are very small, without nucleus or nucleolus. The fibres within the ganglion are often swollen, the medullary sheath is disintegrated, the axone is granular. In other fibres the sheath is full of a disintegrated mass in which the axone cannot be distinguished. In still others the sheath is empty. These changes are shown in Plate III. The connective tissue about the neurone bodies and also about the fibres has been found to be increased in density. The small bloodvessels in the ganglion are sometimes closed by the proliferating tissue in their walls, and all forms of atheroma and sclerosis have been seen in the larger vessels. These changes are shown in Plate VII., taken from Keen and Spiller.[1] Other authors have found marked thickening of the walls of the bloodvessels which lie in the endoneurium of the affected nerves, and this thickening may be present both in the media and intima. In many cases true atheromatous degeneration of the smaller arteries has been found. Similar changes have been found by Thomas in the ganglia on the posterior nerve roots in the early stage of tabes (see Chapter XVII.), and that disease begins with neuralgic pains. In cases of neuralgia attended by herpes, hemorrhages have been found in the ganglia. It is, therefore, probable that neuralgia has, in every chronic case, some organic lesion.

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[1] Keen and Spiller. "On Resection of the Gasserian Ganglion," American Journal of the Medical Sciences, November, 1898.

PLATE VII.

FIG. 1.

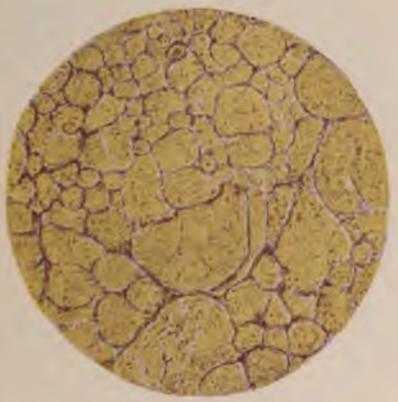


FIG. 2.

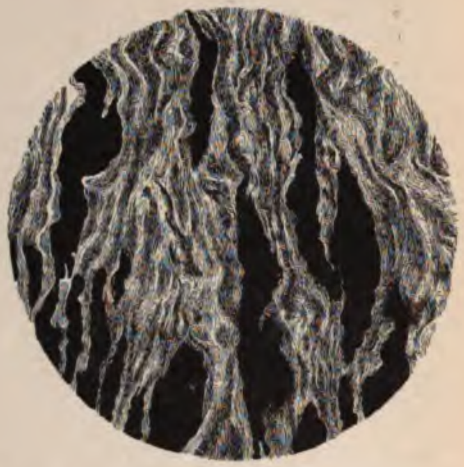


FIG. 3.

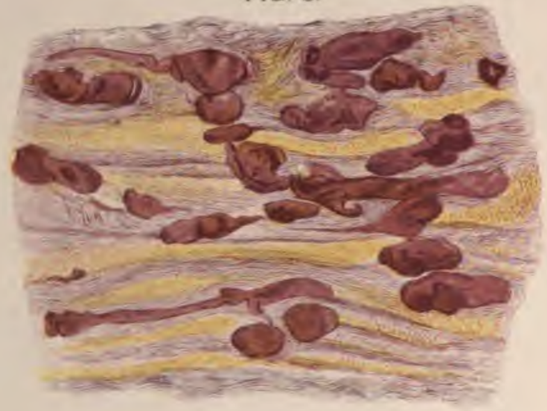


FIG. 4.

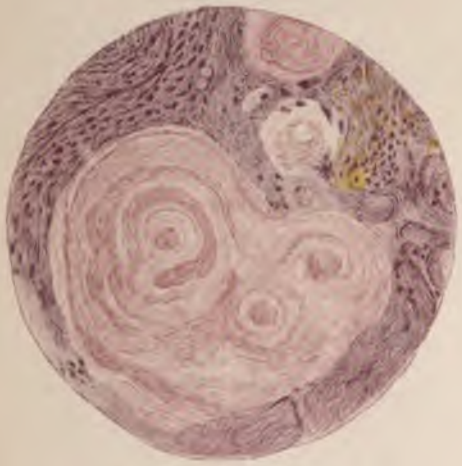
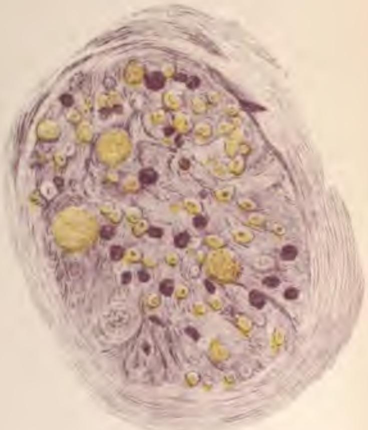


FIG. 5.



While these changes are characteristic of chronic and long-standing cases of neuralgia, especially in cases of trigeminal neuralgia, it is not to be thought that they are present in the acute and transient cases. They are, however, indications of a long-continued malnutrition, and hence, we may conclude that in the acute cases some temporary malnutrition of the sensory neurones is present.

Etiology. Neuralgia may be due to toxic agents of external or of internal origin; organic or inorganic; causing an irritation of the neurones, or of their axones. It may be caused by congestion, without or with a serous exudation in the sheath of the nerve, and consequent compression and irritation of the nerve fibres. It is often due to disease in the bloodvessels accompanying the nerve, which prevents those processes of osmosis necessary to proper nutrition. It is also the first symptom of organic disease in the nerve fibres, or in the endoneurium, of a primary inflammatory character, since many cases of neuritis are preceded by attacks of neuralgia. It not uncommonly develops in nerves which pass through or near organs which are the seat of disease. And it is a frequent manifestation of certain disorders of nutrition, indigestion, stomach or intestinal fermentation, rheumatism, gout, diabetes, anæmia, chlorosis, as well as of the various infectious diseases, especially malaria and syphilis.

The majority of authorities, however, do not regard neuralgia as necessarily a peripheral disease, although its manifestations are peripheral. The sensory nerves send their impulses inward to the gray matter of the spinal cord or brain axis, where such impulses are primarily received in a peculiar substance—the gelatinous substance of Rolando—which appears to be a fine felt-like structure containing many minute cells or nuclei. Thence these impulses are sent up by the central sensory tracts to the cerebral cortex, where they are consciously perceived. An irritation in the gelatinous substance or in the central tracts is capable of producing pain referred to the periphery, as many cases of organic disease in these parts

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DESCRIPTION OF PLATE VII.

FIG. 1.—Portion of the Gasserian ganglion at the entrance of the third branch of the trifacial nerve. The medullary sheaths are most irregularly swollen, and at the right of the field empty nerve sheaths are seen (method of Azoulay).

FIG. 2.—Portion of the second branch of the trigeminal nerve near the Gasserian ganglion. The axis cylinders have entirely disappeared, and the medullary sheaths are greatly swollen. In many places the medullary substance of two or more nerve fibres has united into irregularly shaped masses.

FIG. 3.—One of the nerve bundles within the Gasserian ganglion. Numerous swollen and irregularly formed axis cylinders may be seen. In most portions of the field these appear as drops of a red, hyaline-like substance, but in one portion an axis cylinder of considerable length may be seen.

FIG. 4.—Bloodvessels from the Gasserian ganglion. The walls are greatly thickened, and the lumen of the large vessel has been almost entirely obliterated. In one place the innermost layers of the vessel have contracted from the outer during the process of hardening. Smaller vessels in the upper part of the field are entirely closed.

FIG. 5.—A nerve bundle of the trigeminus close to the Gasserian ganglion. Only a few nerve fibres are present, and everywhere an abundance of connective tissue is seen. Three much swollen medullary sheaths are in the field.

prove. Hence it has been thought that some disturbance in nutrition of the central sensory region is the usual cause of neuralgia, and this theory receives support, as Gowers has held, from the facts that pain may be felt in adjacent parts of several nerve regions, may radiate into adjacent nerve regions, or may be felt in a region not that of the irritated nerve.

Neuralgia may be hereditary. It rarely develops during childhood, but from youth up to old age no one is exempt. Women appear to suffer more than men. It occurs more frequently in nervous and hysterical individuals, and emotional excitement is said to be a cause. It is particularly frequent in sensitive persons in a climate where dampness is common. Attacks bear a distinct relation to atmospheric changes; a low barometer, a high degree of humidity, and a falling temperature combined are often exciting causes.

Certain electric states of the atmosphere, of which as yet we have no precise knowledge, and no means of recording, are capable of causing attacks; and persons who are subject to neuralgia are conscious of sensations which coincide with changes in electrical tension. Thus many persons are warned by a neuralgic attack of the coming of a thunder shower, or of a severe storm, some hours before it arrives. Attacks occur with much greater frequency in winter than in summer. Exposure to cold is the most common cause of an attack, and when a nerve is painful a draught of air often causes a sudden paroxysm, while heat almost uniformly relieves. Patients are usually hypersensitive to cold in the painful area and instinctively protect it when exposed.

Symptoms. The symptom of neuralgia is pain. This is sharp, sudden, shooting along the nerve, and transient, being succeeded by an interval of freedom. The pain is often accompanied by numbness, tingling, or burning in the surface to which the sensory nerve goes; and after a time it is followed by a tender sensitiveness of this part and also of the entire nerve trunk. Pressure on the nerve elicits unusual tenderness and starts an attack of pain. Sometimes the pain is so severe as to cause reflex or automatic spasms in muscles whose motor centres are in close relation to the sensory root involved. Thus trigeminal neuralgia often causes a twitching of the eyes and face; intercostal neuralgia, a bending of the entire trunk; sciatica, a drawing up of the leg. Any movement, especially if sudden, is liable to start a paroxysm of pain, hence patients instinctively keep quiet, move cautiously, and avoid effort. Sometimes vasomotor and trophic disturbances attend or follow a neuralgic attack. Such are flushing of the surface, unusual sweating, or herpetic eruptions on the skin, or falling of the hair. A distinct, unusual pulsation in the vessels of the painful region or nerve can often be felt. A slight swelling of the painful region may also occur.

The course of the disease is very variable. The pain occurs in paroxysms and these vary in severity and duration. The pain may come every few seconds, lasting several seconds, for many hours. Such an attack may recur regularly every day at a definite hour. It often subsides at night, though this is not uniformly the case, and in some patients the pain is worse at night. Attacks rarely continue for more than three or four days when they are succeeded by a free interval of several days, weeks, or even months. Some women suffer from neuralgia with each menstrual period.

The tendency to a recurrence after one attack is well known, and almost everyone is conscious of some nerve in his body of little resistance, liable to give him pain when he is ill A person who has such a tender nerve rarely suffers from neuralgia in other nerves, and neuralgia is rarely bilateral. Occasionally it develops in different nerves at different times. If there is discovered a cause which can be removed, which is rarely the case, treatment of a rational nature can be instituted. But in the majority of cases palliative remedies have to be employed.

Treatment. The causal treatment of neuralgia consists in eliminating all factors capable of producing the disease. These have been mentioned in discussing the etiology. Everything which tends to build up the strength and health of the patient; a life with interests but without anxiety; a healthful regimen with proper diet and exercise; daily baths, either cold or hot, followed by cold sponging; regulation of the digestive functions and of the bowels; washing the kidneys out by drinking an excess of water; and adding to the diet those tonic medicines which occasionally strengthen the nervous system, such as cod-liver oil, quinine, strychnine, iron, arsenic, and the glycero-phosphates will eventually help to cure neuralgia. When any suspicion of infection arises, especially when malaria is detected, a mercurial purge followed by quinine in large doses (20 grains), or by Warburg's tincture (ʒij) (which I consider superior to quinine in many types of neuralgia), will give relief. The quinine or Warburg's extract should be given in large doses four, and again three, hours before the attack of neuralgia is due. In the malarial type a distinct periodicity in the attack is so common that this time can easily be reckoned. A small dose of the drug may be used between the attacks at intervals of six hours. When syphilis is suspected as a cause (and in such cases the attack usually occurs in the afternoon), mercurials, combined with large doses of iodide of potash, are indicated.

In many cases of obstinate neuralgia climatic conditions must be considered, and very often a change of air will be of great benefit. A sojourn at the seashore, or in the mountains, according to the experience of the patient as to the benefit previously derived from travel, will often appear to start a cure. The régime of a sanitarium, or water-cure, as well as the change of air, change of habits and surroundings, and freedom from work or home care, are of great benefit to many patients. And if this can be combined with foreign travel by sending patients to some health resort abroad, much relief will be obtained. The dry climate of the Nile, the rarefied air of the Engadine, or high Swiss valleys, or Colorado, are very beneficial.

The palliative treatment of neuralgia will be considered in discussing the special forms of the disease.

SPECIAL FORMS OF NEURALGIA.

Trigeminal Neuralgia. Neuralgia of the fifth nerve. Tic douloureux. This is the most common form of neuralgia.

Etiology. Women are more subject to trigeminal neuralgia than men. It is a disease which is very uncommon under the age of twenty years, the majority of cases occurring between the ages of twenty-five and fifty years, but no age is exempt, and as the disease is a chronic one, the patients may suffer from it until the close of a long life. I have seen it in patients over eighty years of age. The disease is distinctly an hereditary one, many patients admitting that their parents or grandparents have suffered from it. It develops more commonly in winter than in the summer, which gives support to the theory that the majority of cases are due to exposure to cold. Neurasthenic individuals and those who suffer from anæmia, chlorosis, and gout, or diabetes, are more subject to the disease than others. Head[1] has denied this very strongly, but my observations do not support his statements.

Slight attacks of trigeminal neuralgia may occur in the course of any slight illness, such as a disorder of the stomach, an acute intestinal infection, chronic constipation, congestion of the kidneys, a bad coryza, or angina, or an attack of grippe. In many gouty or rheumatic individuals it is a frequently recurring symptom of the general disorder. Some persons feel a sudden neuralgic pain in the trigeminal nerve while eating ice-cream or drinking very cold drinks. Attacks of neuralgia are not infrequent in connection with migraine. In many persons who are affected with astigmatism, or defective accommodation, neuralgia of the trigeminal nerve may follow any strain of the eyes. Ear disease is also said to have caused it (Moos). Such attacks are extremely temporary and need no special treatment further than the treatment of the causal condition, of which they are the effect. But trigeminal neuralgia may become a more serious and troublesome disease, and such cases require careful study. In chronic lead poisoning it appears to be a not infrequent symptom.

Many cases can be traced to an attack of some one of the infectious diseases, of which malaria, grippe, and typhoid are the most common. In this country when a patient has once had malaria, it is not uncommon for subsequent attacks of this disease to manifest themselves exclusively by attacks of trigeminal neuralgia. Usually such attacks are daily in occurrence, at a definite time of the day, and last for a few hours and disappear as suddenly as they came on. Organic disease of any kind upon the base of the brain, or in the course of the trigeminal nerve, is capable of producing trigeminal neuritis, with a consequent neuralgia. The syphilitic affections of the base of the brain, also tuberculous affections, tumors of the brain, or fractures of the base of the skull, and aneurisms of the internal carotid artery, or of the circle of Willis, bullet wounds of the base of the skull, any disease of the bones of the head and face and catarrhal affections of the nose and throat, dental caries, and any functional disturbance of the eye or ear, are all capable of setting up a neuritis in contiguous nerves and producing either local or trigeminal neuralgia.

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[1] Allbutt's System of Medicine, vol. vi. p. 732.

Symptoms. The chief symptom of trigeminal neuralgia is intense pain in the course of the nerve and upon the face, usually deep, sometimes on the surface. This pain occurs in sharp paroxysms which are repeated frequently, a series of sharp, shooting pains being followed by an interval of relief. Pain is so intense as to be agonizing. It is frequently a burning pain. It makes the patient cringe, it causes a flow of tears, and in very many cases a sharp, quick contraction of the muscles of the face, a closing of the eye and drawing up of the mouth. The patient may even turn the head downward with pain. Changes in the condition of the atmosphere, an increase of the humidity or variations of barometric pressure or of temperature may excite an attack of pain. Some patients have annual attacks during the winter months and are free during warm weather. Any draught upon the face or exposure to cold, any movement of the muscles of the face, the acts of whistling, of talking, of chewing, of swallowing, are capable of arousing the pain, and the patient will very often starve rather than incur the pain of eating. Mental anxiety or intellectual efforts seem capable of starting up an attack. Pain shoots out into the entire distribution of the trunk of the nerve which is affected. In some cases only one trunk is involved, in other cases two trunks, in most severe cases all three trunks. The most common form of neuralgia is the supraorbital, in which the pain is felt above the eye and at the notch or foramen through which the supraorbital nerve makes its exit upon the forehead, and over the forehead and in the hair as high as the vertex. Sometimes the pain radiates into the eyeball, and occasionally pain in the eyeball is the only manifestation of neuralgia of the supraorbital nerve. When the infraorbital branch is the one which is affected, pain is felt upon the cheek and in the upper teeth, and especially in the antrum and malar bone. It may then go as far out as the temple and the lobe of the ear. When the third or lower branch of the nerve is affected, the pain is felt upon the cheek and lip and in the lower teeth, also within the mouth, even in the tongue. I have seen cases in which the neuralgia was limited to the tongue, pain being felt chiefly in the side and lower surface of the organ, being much intensified by the act of swallowing, or talking, or chewing. The pain usually extends in chronic cases from the branch of the nerve first affected into the other branches, so that eventually the entire face is the seat of pain. In one patient a touch along the lower part of the cheek on the right side caused intense pain in the right half of the tongue, and there was also a tender spot over the ramus of the jaw just in front of the ear, but no anæsthesia or loss of taste. A very hypersensitive condition of the skin in the domain of the branch affected is quite frequent. There is a constant sense of tingling, and an unusual appreciation of little variations of temperature; and a slight touch with the fingers, or with cotton-wool is disagreeable and painful. For this reason patients very often instinctively protect the affected part by applications of woollen cloths in order to keep the part at an even temperature and to avoid sudden and unexpected contacts. The face does not often show any change in color, but occasionally it is a little red and the conjunctiva and mucous membrane of the mouth and nose may be congested. Sometimes an increased pulsation of the bloodvessels accompanies the attack of neuralgia and the arteries may be felt to be fuller than usual. It is to be recollected that the most common lesion found in neuralgia is an arterial sclerosis, which may account for the thickening of the arteries that is felt. Sometimes the attack is accompanied by a secretion of tears or of saliva. Trophic changes in the skin of the face have occasionally been observed, the most frequent of which is the appearance of herpes zoster. Some authors have spoken of an unusual grayness of hair and of falling of the beard, but this I have never seen.

Although the fifth nerve conveys the sensation of taste, this is not affected in any way in trigeminal neuralgia—a fact that proves that the taste fibres are independent of those of the fifth nerve proper.

The nerve trunks are extremely tender during an attack of neuralgia at their exits upon the face, namely, over the eye, upon the malar bone, and upon the chin. Any pressure at these points produces very great increase of the pain during an attack. It must be remembered, however, that these points are normally sensitive to pressure. In Plate V. the distribution of the various branches of the fifth nerve in the face is shown.

The course of the disease varies very much in different cases. Usually a neuralgia begins with a sudden attack, which lasts for several days and then passes off under the use of remedies. Such an attack may recur from time to time, and in the severer type the attacks become more and more frequent, so that the patients who have had but one attack in one or two years, after a few years may be having an attack every week. In the most severe type of case the pain comes on every few minutes, day and night, lasting at times for several weeks in this manner, and is quieted only by the strongest opiates, to recur as soon as the effect of the remedy has worn off. It is in the severer type of case that surgical interference is willingly permitted. In any case of facial neuralgia one attack predisposes to another, and patients who have once had an attack appear to be subject to the disease for the rest of their lives. The intervals, however, may be so long (several years) and the attacks may yield so readily to a symptomatic or causal treatment as to preclude any idea of the division of the nerve. I have several patients who never go through the winter without a severe attack. One such patient, who had been a sufferer for many years, escaped while spending the winter on the Nile. In many cases a warm winter climate is advisable. The general deleterious effect of severe pain upon the constitution and nutrition of the patient is often demonstrated in cases of trigeminal neuralgia. The sleep is poor, the assimilation of food is impaired, even where the patients are not prevented by pain from eating; disorders of digestion, especially constipation, are common, and a state of mental disquiet, apprehension, and hopelessness may develop; in fact, some cases have gone on to melancholia and suicide.

The diagnosis of trigeminal neuralgia is very simple; the only question that can arise is whether the neuralgia is based upon an actual neuritis, or is a purely functional disease. The existence of continuous pain, the paroxysms alternating with remissions rather than intermissions, continuous tenderness in the course of the nerves, the development of anæsthesia in the skin of the face, and the determination of some organic disease as a cause, such as syphilis, tumors, etc., mentioned in the section on etiology, make the diagnosis of neuritis rather than neuralgia probable. The facts which are presented in the chapter upon referred pain should not be overlooked in making the diagnosis, and neuralgia should never be confounded with migraine. The pain in migraine is felt in the temple and side of the head, and is not located in the course of the branches of the fifth nerve.

Treatment. The treatment must first be causal; if an organic affection can be ascertained as a basis, it must be treated and removed. In malarial cases and in any case that is of uncertain cause, quinine should be tried. It is well to precede this by a mercurial purge—calomel $\frac{1}{10}$ grain every hour until 1 grain is taken—and to give the quinine in a dose of 20 to 30 grains, which should be administered within an hour and about three hours before the paroxysm of pain is due. Thus the maximum effect of the quinine, which is felt two hours after its ingestion, will coincide with the time of the paroxysm and will prevent its occurrence. While caries of the teeth is an occasional cause of neuralgia, and the condition of the teeth should always be investigated, as the pain may not be felt in the region of the infected tooth, yet in my experience this cause is an infrequent one, and many patients have had all the teeth removed, on the supposition that they were the cause of the affection, without any relief whatever. The so-called Riggs' disease, a dental exostosis to which great importance has been ascribed, is not a common cause of neuralgia, and its treatment is rarely, if ever, followed by relief of the pain, if this is truly neuralgic.

In all conditions of trigeminal neuralgia the general health of the patient must be considered as well as the symptomatic treatment. These patients, as a rule, are anæmic, and are rapidly run down by the starvation that is consequent upon the pain of the act of chewing. It is especially important that every possible means should be used to build up the general health. They should be well fed by means of fluid food, reinforced by nutritive enemata in case solid food cannot be taken. Alcohol, cod-liver oil, beef marrow or medullary glyceride, glycero-phosphate of soda, hypophosphites, arsenic, iron, and strychnine should be used freely as general tonics, and the nutrition generally improved by daily baths and douches and by massage. A change of air is often the one thing that will make the symptomatic remedies efficacious, and the important thing is to secure a change from sea to mountains, or mountains to sea, according to the place where the patient is accustomed to live. In some persons the damp of the sea air tends to produce a return of the neuralgia, and in these persons a dry climate should be sought. A warm climate is essential, and one free from dampness. Egypt, lower Florida, southern California, are all excellent. There are no special baths or health resorts that claim to be efficacious in the treatment of neuralgia, but the régime secured in a foreign water-cure is sometimes of benefit.

Remedies for the pain are very numerous. The most important are the newer analgesics, namely, phenacetin, 10 grains to 15 grains every two hours; acetanilid, 5 grains every two hours; antipyrine, 15 grains every two hours, or salophen, 15 grains every two hours. It is well to combine with each dose of these drugs a grain of caffeine and one-hundredth of a grain of strychnine to avoid their depressing action upon the heart. A hot alcoholic drink sometimes gives immediate relief. Quinine may be used with it.

Aconitine is the best remedy. It is given in pill or tablet form, the French pills of Chapoteaux being the best preparation. Each pill contains $\frac{1}{500}$ grain ($\frac{1}{8}$ milligramme), and the remedy must be used with sufficient frequency to produce constitutional effects. It is my rule to begin with one pill every four hours and decrease the intervals each day one-half hour until one pill every two hours is being given, or until constitutional effects are evident, namely, tingling of the tongue and fingers, sense of general weakness and feebleness of the pulse. It is well to combine with this a fiftieth of a grain of strychnine during the first two days and then $\frac{1}{100}$ of a grain of strychnine, when the aconitine is being given every two hours. If the strychnine does not agree with the patient, or produces twitchings, the dose of this may be reduced, or caffeine two grains, or spartein $\frac{1}{50}$ grain may be given in its place. Some heart stimulant is to be used in connection with the aconitine, when the larger doses are given and the patient should be warned against making any sudden muscular efforts, and during the week or ten days of such treatment should walk very little.

Gelsemium is the remedy next in favor, and this is to be begun in small dose, 10 drops of the tincture, or 5 drops of the fluid extract, being given every three hours, and the dose increased by 1 drop each time until the patient perceives physiological effects, which are a heaviness of the upper eyelids and a difficulty in opening the eyes. In one obstinate case 30 drops of the fluid extract every three hours uniformly cured the attack. This remedy may be kept up at the point which produces this physiological effect for several days and very often gives marked relief. In patients of my own this remedy has succeeded when aconitine had failed completely.

The tincture of colchicum has been used with considerable benefit, especially in the gouty cases. The wine of colchicum may be given in 5-drop doses every two or three hours until a purgative action is produced, and then the dose may be decreased in amount, or the frequency of the dose may be lessened. The effect can be continued for several days.

Butyl chloral hydrate in 5-grain doses every hour for 4 doses has been highly recommended as a remedy for neuralgia of the fifth nerve.

Preparations of arsenic; especially the cacodylate of sodium, $\frac{1}{8}$ to $\frac{1}{4}$ gr., three times a day, are of considerable service. They may be used in conjunction with the remedies already mentioned, or alone, great care being taken to avoid toxic effects.

The most certain and satisfactory remedy is opium or morphine. This may be given in the form of extract of opium, or the tincture of opium in increasing doses, by the stomach, or morphine may be used hypodermically. In the majority of cases of trigeminal neuralgia it will eventually be used, although if possible patients should be prevented from resorting to it too soon, or too freely, as there is no disease in which the morphine habit is a more common sequel than trigeminal neuralgia. Yet in the paroxysms of pain it is imperative to give relief, and the mere fear of engendering the habit should not prevent one from using it in appropriate cases, especially in the early stage of the disease and until other remedies can have the time to exert a constitutional effect.

Strychnine has been used by Dana hypodermically in large doses for the relief of various forms of neuralgia, a dose being begun of $\frac{1}{50}$ of a grain and increased as high as $\frac{1}{10}$ of a grain hypodermically once in twenty four hours. I have seldom seen any benefit result from this treatment, but I have seen severe strychnine poisoning ensue.

In individuals who are the subject of arterial sclerosis nitroglycerin is a remedy of considerable efficacy, which may be used in $\frac{1}{100}$ or $\frac{1}{150}$ of a grain and repeated with sufficient frequency to produce a perceptible effect upon the arterial tension. Nitrite of sodium in 1 or 2 grain doses is equally efficacious.

The extract of cannabis indica has some reputation in the treatment of neuralgia. It is to be given in tablet or pill form, $\frac{1}{4}$ of a grain at a dose several times a day.

Preparations of the bromides, and bromide combined with chloral may be of some service in the lighter forms of neuralgia, but in the severe attacks do not have sufficient effect to quiet the pain. If a small amount of morphine be added to full doses of these two drugs a useful remedy is obtained.

Local applications to the face sometimes give considerable relief. Applications of ice bags or freezing the face by a spray of chloride of ethyl applied to the point of exit of the branch of the nerve affected are sometimes of considerable service. The majority of patients, however, prefer hot applications; and a hot-water bag or a poultice or an application of cotton covered with oiled silk, outside of which a hot bag or a Japanese hand furnace can be placed, will often give considerable relief. Most of these patients prefer to protect the face by some woollen substance and thus avoid exposure to cold or to changes of temperature.

Sometimes local applications of lotions and salves are of service. Weak chloroform liniment, evaporating lotions of opium and lead, or ointments containing aconite, morphine, atropine, and veratrine, have been used with some success. Rubbing the face with a menthol pencil may give relief. The face may be painted with collodium in which iodoform, 1 to 15 grains, has been dissolved, a thick coating being applied over the exit of the branch of the nerve affected: Camphor and chloral rubbed together into a paste and applied has been of benefit.

Inhalations of chloroform may be given with good effect, but this, of course, must be used with great caution.

Electricity in the form of galvanism, a mild, continuous current, with the positive pole upon the painful point and the negative on the back, has been recommended. The application should be five minutes in duration over each branch of the nerve and great care should be taken not to make or break the current during the application, it being begun very gradually by the aid of a rheostat. Sometimes a severe vertigo may be produced by applications of galvanism to the head and face. I have never seen any good result from this method of treatment.

Massage of the face is usually very painful in conditions of neuralgia and has never in my experience been of much service in the treatment of the attacks. Vibrations maintained by a tuning-fork electrically vibrated, the end of the fork being in contact with the affected branch of the nerve, and vibrations with the end of the fingers of an expert masseur, have given relief in some cases.

The actual cautery cannot be applied to the face on account of the scars which remain, and hence is not used in trigeminal neuralgia.

Surgical Treatment. For many years division of individual branches of the nerve has been practised for the relief of neuralgia, the nerve being merely divided or being divided and the central end stretched, or a considerable section of the nerve being removed.

To reach the peripheral parts of the supraorbital or infraorbital nerves, semilunar incisions of the eyebrow or cheek are made in the natural lines of the skin, and chiselling out of the orbital bones gives access to the nerve. Division of the third branch of the nerve within the mouth, as it passes into the ramus of the jaw, is a comparatively simple operation within the reach of almost any surgeon. The deeper operations of division of these various roots at their exit from the skull requires much greater surgical skill and is very complicated and bloody.

While such operations may give relief for some months or years, they are not positively curative, but they may be performed when the case is a chronic one and the neuralgia is limited to a single branch of the nerve. In the majority of cases the neuralgia eventually extends to other branches, and in such cases the more radical operation advised by Krause and Hartley simultaneously, of exsection of the Gasserian ganglion within the skull, may be performed. This operation is carried out by making a horseshoe-shaped incision through the skin, periosteum, and temporal and parietal bones, and laying down a flap which should be at least four inches in diameter, the base being half an inch above the base of the skull. The dura is not incised, but with the brain is then carefully lifted away from the base of the skull. The point of exit of the fifth nerve is determined, great care being taken to avoid injuring the optic and oculomotor nerves; the free branches of the nerve are seized, the Gasserian ganglion is pulled outward through the dura, and the trunk of the nerve divided behind the ganglion. Traction on the nerves is made and they are divided as far as possible from the ganglion, which is thus removed with a half-inch of the nerves. The result of this operation is to deprive the face entirely of sensation, and the operation is always followed by a permanent condition of tingling and numbness, with anæsthesia of the face, and loss of taste on one side of the tongue; but it usually gives permanent relief from the neuralgia. The agonizing pain is so great that these patients are willing to submit to any operation that promises a cure. I have seen a large number of permanent cures in patients of my own upon whom Hartley has performed this operation, and Keen, Krause, Horsley and others have published many cases of cure. Recently, Frazer and Keen have obtained good results by dividing the central end of the nerve just behind the Gasserian ganglion, without removing the ganglion.

Cervico-occipital Neuralgia. Neuralgia of the posterior branches of the cervical plexus and especially of the great occipital nerve, which passes up the back of the neck and over the back of the head to the vertex, is occasionally met with, though it is by no means as common as trigeminal neuralgia. Pain is felt along the course of the nerves in the neck, but particularly below the occiput and in the vertex. There are painful spots near the exit of the nerve at the base of the skull, over the occiput, and upon the vertex. The scalp is very likely to be tender when the pain is intense. Frequently the pain is bilateral, and very often is continuous rather than paroxysmal, though sharp attacks of pain may follow any motion of the head. The patients usually hold the head in a fixed position to avoid the pain produced by movement. Sometimes a swelling of the cervical glands accompanies the neuralgia. In one case reported by Johnson, the superior ganglion and its cord were fixed by adhesions which were freed by operation, after which the patient was relieved of the neuralgia. Occasionally an attack of trigeminal neuralgia is associated with the cervical neuralgia. This may be due to the central radiation of irritation, which is erroneously referred, or it may be due to a common cause producing both affections. The characteristics of the disease are similar to those of trigeminal neuralgia and the treatment is that of neuralgia in general. In one very obstinate case under my care, McBurney divided the great occipital nerve at its exit from the muscle. This had only a temporary effect and it was several months before a spontaneous recovery ensued.

Brachial Neuralgia. The various nerves of the brachial plexus are occasionally the seat of neuralgia. The pain may be felt chiefly in and about the plexus in the neck, but usually shoots outward into the branches down the arm and into the forearm and hand. There are painful points where the nerves are superficial and can be easily compressed against the bones. Thus in the axilla, over the circumflex, near the deltoid, over the musculospiral as it curves about the humerus, over the ulnar at the elbow, and over the nerves at the wrist, pressure causes pain. The pain is made worse by any movements of the arm, and the patient instinctively keeps it quiet. The pain is usually worse at night, probably because of pressure exerted during sleep, and hence sleep is often interfered with. There is often much hypersensitiveness of the skin and a burning pain, which Weir Mitchell has named "causalgia." Herpes zoster is a frequent complicating symptom, and the position of the vesicles corresponds exactly with the distribution of the cutaneous branches of the nerves. Glossy skin and trophic changes in the nails appear only when a true neuritis has developed.

Intercostal Neuralgia. While the intercostal nerves from their protected situation are usually exempt from injuries and wounds, excepting in cases of stab wounds of the back or chest, they are frequently the seat of neuralgia. The neuralgic pain is sharp and shooting around the side of the chest and is often felt chiefly in the terminal filaments of the nerve in front or at its middle branch in the line of the axilla. At these two points of exit of the branches and also at the point of exit of the posterior branch near to the spine, points of tenderness are usually present. If the upper dorsal nerves are affected the pain may also be felt in the inner side of the arm and may be attended by tachycardia. The pain is increased by breathing, by coughing, or by any movement of the chest. A dull, aching, pressing pain is felt between the attacks, and patients usually like to support the side and compress it. Others cannot endure the touch of their clothing. Intercostal neuralgia is very often followed by an attack of herpes zoster. The cases of Kaposi have proved that when herpes occurs the disease is sometimes due to a hemorrhage within the posterior spinal ganglion of the nerve affected. In other cases, a parenchymatous and interstitial neuritis have been found. The herpes may precede the neuralgia and the latter—especially in old people—may persist long after the eruption has subsided.

Any of the ordinary causes of neuralgia may produce intercostal neuralgia. Intercostal neuralgia is frequently due to a secondary implication of the nerve from disease, either in the bones of the spine or chest, or in infectious conditions of the pleura. Thus empyema is occasionally complicated by intercostal neuralgia. It has occurred from pressure by aneurisms. Its relative frequency in women suggests that the same causes that produce mastodynia (*q. v.*), a variety of intercostal neuralgia, may also cause the disease.

The **diagnosis** of intercostal neuralgia is made from the exceeding sharp, shooting character of the pain, and by the existence of painful points upon the chest at the exit of the nerves. It may suggest angina pectoris when tachycardia occurs, but the course of the two diseases differs so widely that a diagnosis is easily reached.

Pleurodynia, which is a muscular rheumatism of the intercostal muscles, produces a somewhat similar pain, increased by respiration; but in this case the tenderness is not felt especially at the point of exit of the nerve. In pleurisy the pain is less exactly limited in its distribution, and the physical signs of the disease enable a diagnosis to be reached.

The **treatment** is by local applications along the course of the nerve; blisters or the actual cautery over the exit of the spinal branches, and special measures such as are described in the treatment of neuralgia in general. Edinger recommends a spray of chloræthyl. For the relief of the herpes, applications of oxide of zinc ointment may be made, or the herpetic vesicles may be painted with collodion, to which a small amount (1 per cent.) of carbolic acid or thymol has been added. Care should be taken to avoid any septic infection of the exposed skin when the vesicles have ruptured. In old persons opiates should be very carefully given.

Neuralgia of the Breast—Mastodynia. This form of neuralgia is very rare and occurs chiefly in women. It may develop during pregnancy and is especially frequent during the last weeks. It occasionally occurs during lactation and may be so severe as to interfere greatly with nursing. It sometimes develops in women during the menstrual period, or in the course of uterine or ovarian disease. In one of my cases the attacks lasted three days at each monthly period for several years, requiring the use of morphine at the time. It also occurs as a direct result of carcinoma and other tumors of the breast; but occasionally it occurs as a result of anæmic or neurasthenic states without concurrent affection of the genital organs. In fact, any one of the general causes of neuralgia may be capable of producing mastodynia.

The pain is usually located deep in the gland, is very severe, and occurs in paroxysms, but there is usually a dull, heavy feeling or aching pain all the time. Sometimes the pain is superficial, and the skin of the breast, especially about the nipple, is exquisitely sensitive; the nipple may be congested and the entire breast red and swollen. Erb has seen a case in which the pain excited the function of the gland. There are tender points along the spinous processes of the second to the sixth dorsal vertebræ. The course of the case is a slow one. Sometimes the pain persists in spite of treatment until pregnancy is over, or until lactation ceases.

In addition to general measures of treatment for neuralgia already described, some relief attends the adjustment of a support to the breast, or a firm bandage to the breast. Local applications of cocaine in 4 per cent. solution to the nipple sometimes quiets the pain. Ointment of belladonna and aconite or evaporating solution of lead and opium may be of benefit. Nageli has afforded relief by stretching the nerve, the entire breast being firmly grasped in both hands and slowly but steadily lifted for twenty or thirty seconds several times a day. This is a simple method which should be tried in every case.

Lumbo-abdominal Neuralgia. The lower dorsal nerves and the lumbar nerves send branches over the back and abdomen. These branches are occasionally the seat of neuralgia, which resembles intercostal neuralgia, to which it is strictly homologous. The pain shoots about the side of the body and is at times intense and often burning in character. There are tender points at the posterior, lateral, and anterior exits of the branches of the nerves. Neuralgia in this locality is especially likely to be attended by herpes. I have seen a case in which abdominal neuralgia followed intercostal neuralgia, both being attended by herpes. In this case, as in many others, the course of the case was slow, the pain lasting several weeks, and resisting all forms of treatment. The patient was a middle-aged woman, the subject of chronic arterial changes and cirrhosis of the kidneys. The pain in lumbo-abdominal neuralgia is often dull and continuous. It frequently extends downward into the groin and is felt in the penis or labium, on which parts herpes appears. This condition is not to be confounded with neuralgia of the testicles and ovaries, which is an affection of the sympathetic system supplying these organs.

There are no special therapeutic indications in this form of neuralgia.

Crural Neuralgia. The anterior crural nerve, which extends down the inner surface of the thigh and leg and reaches the ankle, is occasionally the seat of neuralgia. The condition may develop in the course of sciatica. It is more common in men than in women, and usually develops after hard muscular work or long marches, exposure to cold, or injuries. It is to be remembered that pain in this nerve is often the first sign of caries of the spine. It is said that in diabetes crural neuralgia and sciatica are the most frequent forms of neuralgia produced. The most obstinate case of the disease which I have seen was in a diabetic patient. Painful points are found in the inguinal canal, on the front of the thigh, on the inner side of the knee, and on the inner malleolus. Herpes frequently appears. This form is not to be mistaken for the reflex pain often felt by women at the menstrual period or with ovarian disease in this locality. The treatment is the same as that for sciatica.

Sciatica. Sciatica is neuralgia of the sciatic nerve. It is a very common affection, more frequently met with in males than in females, and in adults than in persons under the age of twenty years. Persons between the age of forty and fifty years are most liable to the disease.

Etiology. Gout and rheumatism are the chief causes of sciatica. It can often be directly traced to exposure to cold. It is particularly frequent in diabetic patients. It may follow any of the infectious diseases or may be due to alcoholism. The poisonous agents which cause multiple neuritis may cause sciatica. Direct pressure upon the sciatic nerve by sitting in an uneasy position or in hard chairs with sharp edges, or pressure exerted from causes acting within the pelvis, such as accumulations within the rectum, pelvic inflammation, or tumors of the uterus or ovaries, or the condition of pregnancy may cause sciatica. Disease of the sacral or thigh bones may produce sciatica. In one case under my care a long-standing sciatica was finally explained by the development of an osteosarcoma of the pelvis. Injuries are a frequent cause of sciatica, as in falls upon the buttocks and fractures of the thigh. Lifting heavy weights has brought on sciatica. Workers who stand or move the legs at machines are particularly liable. The sciatic nerve is well supplied with bloodvessels, and it is possible that a venous congestion from pressure is an exciting cause of disturbance of function in the nerve in these cases. Sciatica often follows hemorrhoids and varicose veins in the leg. It is difficult to separate sharply sciatica from sciatic neuritis, and in many cases where the disease lasts a considerable length of time, it is reasonable to suppose that a neuritis is present. In fact, in all the cases in which an autopsy has been obtained, an interstitial neuritis with congestion of the vessels, hemorrhages in the sheath, and secondary degenerations in the nerve fibres have been found. Many cases are so short in their duration, the symptoms are so distinctly intermittent, and the cessation of the pain is so instantaneous either upon change of position or upon local applications, as to make it improbable that a true neuritis has developed.

The **symptoms** of sciatica are pain in the back of the thigh and in the outer side of the leg, referred quite distinctly to the course of the nerve. Sometimes when the upper branches of the nerve are involved, pain is felt over the sacrum and buttock as high as the waist-line. In other cases these branches escape. Usually the pain is most intense about the sciatic notch and down the back of the thigh to the knee. Sometimes it is limited to the lower branches of the nerve upon the outer side of the leg to the foot.

In the severer type of case the entire distribution of the nerve is the seat of pain. The pain is not as diffuse as in muscular rheumatism, and when the patient is asked to indicate its position he follows down with the tips of his fingers the line of the nerve from its exit in the sciatic notch to the external condyle of the ankle. The pain occurs in paroxysms and may be agonizing in its intensity; is usually increased by movement, by walking, especially by going up stairs, and can always be elicited by hyperextension of the leg upon the thigh and of the thigh upon the pelvis—a position which stretches the sciatic nerve. It is attended by tenderness at those points where the nerve can easily be compressed between the fingers and a prominent portion of the bone, namely, above the hip-joint near the posterior iliac spine, at the sciatic notch, at the middle of the thigh, behind the knee, below the head of the fibula, behind the external condyle of the ankle, and on the back of the foot. The pain is usually dull in character and constantly present, but there are acute exacerbations and these are often attended by sensations of burning or feeling as if water were trickling along the limb, or sharp, darting pains through the length of the nerve. The pain is a deep-seated one and is referred to the muscles or the bone, never to the skin. It may be increased by extension of the leg and thigh together. Pains are usually worse at night, when the patient gets warm in bed, though, as a rule, warmth to the limb is grateful and the patients instinctively avoid exposure to cold.

The gait is affected in sciatica, the patient limping and moving the entire limb stiffly. The toe is turned out, the leg is slightly abducted, and the patient instinctively rotates the spine to the well side. In extreme cases there may develop a tendency to lateral curvature of the spine away from the affected side. In the more intense cases the patient is confined to the bed by the pain, cannot bear his weight upon the limb, cannot bear to have it moved, and finds it easy only in one position, which varies in different cases. The leg is usually abducted, rotated outward, and flexed at the knee. It is best to allow the patient to ascertain the position in which he is most comfortable and to keep the limb in that position supported by pillows. The pain may be so intense as to cause reflex spasms and twitchings of the leg, and is very commonly associated with tingling and numbness in the leg, or by sensations of pins and needles, the foot being asleep constantly. When the sciatica goes on to a neuritis, anæsthesia may develop in the outer side of the leg below the knee and in the back of the foot, and the muscles of the calf of the leg may be weak or even paralyzed and atrophied, showing a reaction of degeneration

The course of the disease varies very much in different cases. There is sometimes a sudden, acute onset which in cases due to exposure to cold or to rheumatism, or to gout, is often attended by a slight rise of temperature and constitutional disturbances. Usually the onset is gradual. Pain occurs in paroxysms, which become more and more frequent and severe during several days, and by the end of a week the pain has become continuous. In bad cases the patient limps or is unable to walk. This condition may remain for several weeks or may subside gradually. Recovery ensues within three or four weeks, though in many cases months pass before the patient is free from pain. There is a great tendency to relapse in cases of sciatica. I have rarely known a patient to escape a second and third attack. Even when recovery has ensued, pain can be produced by the slightest tendency to overstretch the nerve, or by too great exertion in walking. I have never seen a bilateral sciatica, though a number of cases are recorded. Even in the chronic cases improvement after a time occurs, but where a neuritis has been set up the pain is remittent rather than intermittent. In the cases which are due to pressure within the pelvis the pain is more likely to be in the periphery of the nerve and its terminal distribution than in the trunk upon the thigh. In the cases where neuritis is present there is usually tenderness along the entire course of the nerve, as well as at the painful points of Valleix.

Herpes is a common complication of sciatica. It is sometimes very extensive over the back of the thigh and on the buttock. The herpetic vesicles may become large and confluent, so that the affection of the skin may give as much discomfort as the pain. In this condition the skin should be protected by applications of oxide of zinc ointment and bandaging with cotton-wool.

French authors have called attention to the characteristic position of the limb in standing and in walking, which is characteristic of sciatica and serves to distinguish it from muscular rheumatism, or from hip-joint disease.[1] In sciatica the thigh is somewhat adducted, the fold of the buttock is depressed, and the knee is slightly flexed, even when standing in an upright position. The entire spine is somewhat deflected with convexity to the affected side. This is due to an instinctive effort to support the weight on the well limb There is sometimes a slight wasting of the muscles in the limb.

The **diagnosis** of sciatica is usually evident from the characteristic symptoms, but it has been mistaken for disease of the hip-joint, for caries of the sacrum, and for lesions in the cauda equina. In hip-joint disease the pain is usually felt first in the inner side of the knee. It is not associated with tenderness along the course of the sciatic nerve, though pain may be felt all about the hip-joint. There is usually a limitation of the motion of the joint and possibly some shortening of the thigh, and a characteristic rigid position of adduction with slight rotation is found in hip-joint disease. In caries of the sacrum, while the pain may be felt in the sciatic nerve, there are usually symptoms of tenderness along the sacrum, tenderness in the motion of the pelvic bones upon the sacrum, and the pain is less unilateral than in sciatica. A rheumatic affection of the sacro-iliac tendons and ligaments does not produce pain below the exit of the sciatic nerve in the thigh. In muscular rheumatism the pains are diffused and are not located exactly in the course of the sciatic nerve.

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[1] See also H. Ehret. Mittheilungen aus der Grenzgebiet der Med. u. Chirurg., 1898, p. 695.

In affections of the cauda equina, meningeal thickening, hemorrhages or tumors the symptoms are rarely, if ever, unilateral in character. Considerable pain is usually felt over the sacrum, and incontinence of urine and feces, with anæsthesia about the rectum, are early symptoms which are not present in sciatica.

Locomotor ataxia very often produces severe pains in the course of the sciatic nerve, and although the earliest sign of this disease is more often pain in the distribution of the crural nerve, sciatica may be its first symptom. In every case of sciatica, therefore, the knee-jerk should be tested and the pupil be carefully observed to detect any lack of reaction to light; these reflexes are not affected in sciatica. Anæsthetic areas should be sought for, as these are not present in true sciatica, but develop early in locomotor ataxia.

The **prognosis** in sciatica is fairly good as regards eventual recovery, but no statement can be made with regard to the duration of any particular attack, for although the duration of each attack is somewhat in proportion to the severity of the symptoms, relapses are so common that statements in regard to duration are never precise. I have known cases to last two or three years and then recover.

Hyde has given some interesting facts in his analysis of 200 cases which may be quoted here. The duration was less than one month in 45 cases; from one to three months in 70 cases; from three to six months in 29 cases; from six to twelve months in 29 cases; two years in 15 cases; three years in 6 cases; four years in 1 case; five years in 2 cases, and six, seven, and ten years in one case each. Forty-three of Hyde's cases had had previous attacks.

Treatment. In addition to the general statements with regard to the treatment of neuralgia, all of which may be applied to sciatica, a few special indications may be given. The cause must be removed if possible. In all cases active purgatives must be given at the outset. In cases due to cold or rheumatism salicylates and salophen are of the greatest service. Salophen may be given in 15-grain doses every two hours. Absolute rest of the limb is most important, and the patient should be advised to remain upon the bed or couch, even from the beginning of the disease, being allowed to assume the position which gives the greatest comfort, as this varies in different individuals. All positions and all movements which intensify the pain should be avoided. I have not seen any benefit from fixation of the limb, either by a long splint, as has been recommended by Hammond, or in a plaster bandage as has been suggested by others. Hot applications are of the greatest service both in the acute and chronic stage of the disease. The old-fashioned remedy of ironing the limb with a hot iron is of distinct service. The application of flaxseed poultices is of benefit in the acute stage, but the most efficacious remedy is the application of the Paquelin cautery along the course of the nerve. This should be applied daily from the very beginning of the disease. Or small fly-blisters should be applied at the points of tenderness along the trunk, and should be repeated every other day. If the cantharides is applied by means of collodium the blisters need not be more than one-quarter of an inch in diameter, and as many as twenty may be placed in the course of the nerve.

A very efficacious method of the treatment of sciatica is exposure of the limb to very high temperature in an apparatus in which hot air can be produced. Thus the temperature of the air may be raised to 250° or 300° F., the limb being encased in the apparatus. And such applications may be repeated twenty minutes in duration daily.

It often gives much relief to expose the limb to a stream of steam along the course of the sciatic nerve. Hot-water massage is also of a great deal of service, such as is applied at Wildbad, Ragatz, and Bath, at Aix-les-Bains and at the Hot Springs of Virginia, the stream of water under pressure from twenty to thirty, or even forty pounds, being directed upon the limb, the painful points along the nerve being protected by the hand. Long-continued hot sitz baths are often of service and give the patients relief. Richfield Springs has a good reputation in the treatment of sciatica, and I have known patients to recover after a course of sulphur baths there. Massage of the limb is sometimes of service if it is done with great skill and pressure is but lightly exerted upon the nerve trunk. Mud or sand baths heated to 100° F. are of great service. A spray of chloride of ethyl which causes local freezing sometimes gives immediate relief. Hypodermic injections into the nerve have been practised and recommended. Hypodermically an injection of chloroform, of a 2 per cent solution of cocaine, or of a 1 per cent. solution of atropine, or simply of distilled water, may be given. Osmic acid solutions are dangerous. In a few cases where the nerve has been felt to be much swollen puncture by a needle has resulted in the evacuation of serum from the sheath, and has given relief. Long needles are to be used under strict aseptic precautions. They are to be inserted and left in an hour, the limb being kept immovable during this time. Liniments and ointments containing belladonna or aconite may be rubbed in along the course of the nerve, and are sometimes of benefit. The general medicinal treatment by internal remedies may be conducted along the line already described in the treatment of neuralgia. Electricity I have never found of much service, but long-continued applications of galvanism of very mild strength (five or six milliampères) from large sponges may be tried.

In chronic cases where all other means have failed to relieve it has been proposed to stretch the nerve, and this is probably of service in cases where an interstitial neuritis has produced adhesions between the adjacent bundles of nerve fibres and constriction of the blood-vessels. A nerve may be stretched by using hyperextension of the leg, a proceeding which has to be done under chloroform, as it is very painful. Or the nerve may be stretched by cutting down upon it just below its exit in the sciatic notch, dividing the sheath and stretching it with the fingers. Some cases have been reported of improvement after this procedure. I have seen one cure in a very obstinate case. I have seen several failures. Other cases have been reported in which nerve stretching has been followed by paralysis in the muscles supplied by the affected nerve. It should only be resorted to after every other means has been tried, and no positive promise of relief should be given.

Neuralgia of the Testicles or Ovaries. Neuralgia in these organs is usually the result of some congestion of the veins due to organic disease or to pressure. But occasionally it may be due to some one of the general causes of neuralgia already mentioned. The pain is very intense and most demoralizing. It causes great restlessness and much mental depression. It is occasionally attended by sexual excitement. Neuralgia of the ovaries may coincide with the menstrual function.

The treatment is first directed to reducing local congestion. This may be done by elevating the pelvis and letting gravitation empty the veins. Applications of heat, especially hot poultices to the testes, and hot vaginal douches or rectal injections, to be taken in a recumbent position, are usually of great service. Resort may be had to the stronger narcotics early, as it is better to cut an attack short than to allow it to go on. And attention to general measures for building up the health must not be omitted.

Neuralgia of the perineum, of the prostate gland, and of the neck of the bladder in males, and neuralgia of the rectum in both sexes, is occasionally complained of. If no local disease is present and the affection is not traceable to lithæmia or to hemorrhoids, it is probably caused by local congestion. Massage of the perineum, the pelvis being raised, will give relief, or hot applications to the perineum may be effective. If these fail, massage of the prostate through the rectum, or stripping the seminal vesicles may do good. A continuous irrigation of the rectum with hot water by means of the double tube devised by Keyes is of much service in these cases. Sitz baths, either hot or cold, are of value. As in neuralgia of the genital organs prompt relief must be given by narcotics to avoid a state of mental depression. I have found an ointment or suppository of cocaine and hyoscyamus of great service. Weir Mitchell has seen this form of neuralgia as an early sign of tabes, an observation which I can confirm, having seen two such cases. I have also known it to occur in lithæmic individuals and in persons whose circulation was poor, after sexual excitement, both of a natural and of an unnatural kind.

Coccygodynia. Neuralgia of the coccygeal nerves is an exceedingly rare affection. But pain in the tip of the spine is very common, and has received the name of coccygodynia. It may be a local affection, due to injuries, especially in women after confinement; to falls on the seat or to caries of the spinal bone. It is usually in my opinion a referred pain of central origin, and may develop in very many functional or organic diseases of the nervous system. In neurasthenic and anæmic persons, chiefly in women, it is a frequent complaint. It may be associated with pain in the nape of the neck or between the shoulders, with irritable spine, or with any hysterical state. It is a very common complaint in cases of traumatic neurosis. In the majority of cases it is a purely hysterical symptom, and is increased by any local treatment or by anything which directs attention to it. In several such cases where gynecologists have exsected the coccyx the pain has persisted after the operation. As the hysteria or neurasthenia passes off the pain is forgotten. Hence it should never be treated directly, but only as a symptom of a general condition. In the few cases which are true neuralgia the same treatment as that of sciatica will be applicable.

REFERRED PAINS.

There are a great many conditions of disease in which more or less severe pain is felt in various parts of the body at a distance from a diseased organ. The explanation for the referring of these pains to a part of the body which is really not affected is as follows:

The branches of the visceral nerves and of the general sympathetic nervous system enter the spinal cord at various levels throughout its entire length. Irritation sent into the spinal cord through these nerves set up sensory impulses in the various segments of the cord, each segment receiving impulses from a certain organ. These sensory impulses are sent upward to the brain, and become conscious perceptions. They are referred by consciousness not to their actual point of origin, but to the part of the body from which sensations usually come when received at the particular segment irritated. Thus, as in general experience, sensations and pains coming from the various segments of the spinal cord have been due to irritation in the surface of the body corresponding to these segments; these various visceral sensations are referred to the surface of the body. Some examples of such referred pains will make this matter clearer.

It is not at all uncommon in eyestrain to have a pain felt in the forehead or in the back of the neck, neither of which parts is in direct connection with the eye.

The pain produced by decayed teeth may be felt in the temple or behind the ear, instead of in the jaw.

Severe pain in the back of the head is a common symptom of uterine disease or of inflammation of the bladder.

Pain down the left arm is a common symptom of heart disease, and may be attended by hyperæsthesia in the region of the fourth and fifth dorsal nerves on the chest.

Pain in the wrist on the flexor surface is frequently felt in disease of the uterus, ovaries, or bladder.

Pain under the right shoulder-blade is frequently felt in disease of the liver, and is often attended by hyperæsthesia in the domain of the eighth to the twelfth dorsal nerve.

Pain under the left shoulder-blade is common in enlargement of the spleen.

Pain between the shoulder-blades is a very common symptom of gastric affections of any kind. It may be attended by hyperæsthesia in the epigastric region, and the nearer the disease to the cardiac end of the stomach—*e. g.*, ulcer—the higher the pain is felt. In severe vomiting pain may be felt on the back of the arms or even down the back of the forearms.

Pain across the small of the back is common in colitis or in impaction of feces within the colon.

Pain across the upper sacral region is very common in uterine disease.

Pain over the outer side of the hip is usually due to ovarian congestion.

Pain down the inner side of the leg is also due to the same cause.

Pain on the inner side of the knee is an early symptom of hip-joint disease.

Pain in the heel is a frequent symptom in lithæmia, and may also be felt in ovarian disease.

Dana[1] has studied the location of these reflex or referred pains very carefully, and his figures (Fig. 26), which are here reproduced, demonstrate the areas of pain of sympathetic origin.

Head[2] and Mackenzie[3] have added a few facts to Dana's statements.

There is hardly any viscus disease in which may not give rise to some of these referred pains, and it is evident that without some knowledge of the localities of these referred pains the presence of such pain might mislead the physician. I have had many patients brought to me for supposed spinal disease because of pain in the dorsal region and tenderness of the dorsal spine, with pain in the epigastrium, general weakness and neurasthenic conditions, and increased knee-jerks, who have been relieved by the correction of a chronic gastric dyspepsia.

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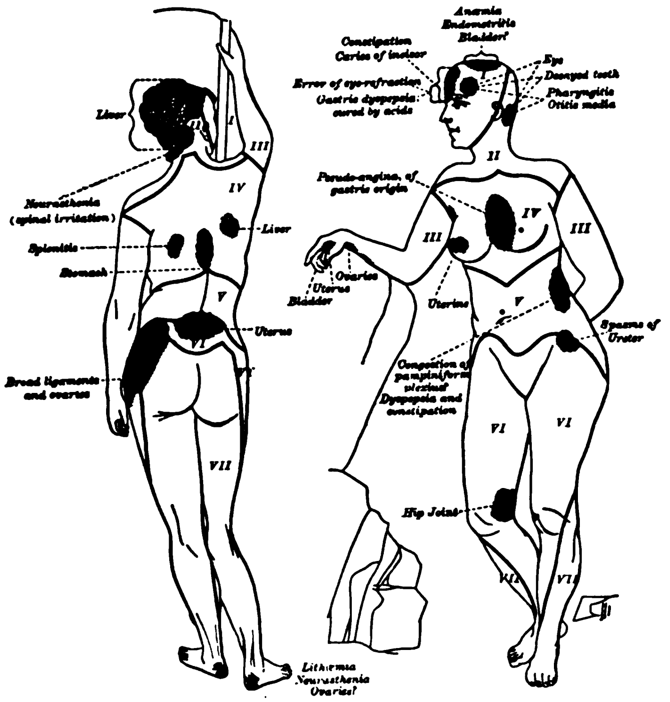
[1] New York Medical Journal, July 23, 1887.

[2] Brain, 1893, 1894, 1896.

[3] Brain, 1902.

It is evident that in these referred pains the treatment must be directed to relief of the functional or organic disease in the viscus which is affected. It may be added that not infrequently sharp counter-irritation over the seat of pain is followed not only by relief of the pain, but also by improvement in the visceral condition which is its cause.

FIG. 26.



The location of referred pains and their cause. (Dana.)

Area.	*Cerebro-spinal nerves.*	*Distribution.*	*Associated ganglia of sympathetic.*	*Distribution.*
I.	Trigeminus, facial.	Face and anterior scalp.	4 cerebral.	Head.
II.	Upper 4 cervical.	Occiput, neck.	1st cervical.	Head, ear.
III.	Lower 4 cervical and 1st dorsal.	Upper extremity.	2d and 3d cervical, 1st dorsal.	Heart.
IV.	Upper 6 dorsal.	Thorax.	1st to 6th dorsal.	Lungs.
V.	Lower 6 dorsal.	Abdomen, upper lumbar.	6th to 12th dorsal.	Viscera of abdomen and testes.
VI.	12th dorsal and 4 lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.	1st to 5th lumbar.	Pelvic organs.
VII.	5th lumbar and 5 sacral.	Lower gluteal, posterior thigh and leg.	1st to 5th sacral.	Pelvic organs and legs.

CHAPTER V.

MULTIPLE NEURITIS.

Varieties. Etiology. Symptoms. Course. Duration. Diagnosis. Prognosis and Treatment.

MULTIPLE neuritis or polyneuritis is a general disseminated inflammation or degeneration of the nerves, symmetrical in its distribution upon both sides of the body and generally affecting all the nerves of the limbs, particularly in their terminal branches. It is hence called peripheral neuritis. The affection rarely, if ever, extends as high as the nerve plexuses.

Varieties. There are a number of forms of multiple neuritis which differ from one another somewhat in their pathology and in their symptomatology. The following etiological classification of these various forms is of practical use.

1. Toxic cases due to the action of a poison derived from without the body. These poisons are alcohol, carbonic oxide gas, bisulphide of carbon, the coal-tar products, especially sulphonal and trional; and nitrobenzol; also arsenic, lead, mercury, copper, phosphorus and silver.

2. Infectious cases due to some agent acquired or developed within the body as an accompaniment or sequel of diphtheria, grippe, typhoid, typhus, malaria, scarlet fever, measles, whooping-cough, smallpox, erysipelas, and septicæmic conditions, including gonorrhœa and puerperal fever, epidemic forms of beriberi or kakke, and leprous neuritis.

3. Cases due to general diseased states of the body whose origin is undetermined, such as rheumatism, gout, diabetes, anæmia, marasmus, general malnutrition consequent upon tuberculosis, syphilis, and senility, carcinoma, and local malnutrition produced by arterial sclerosis.

4. Cases due to exposure to cold and developing spontaneously without known cause.

Etiology. Multiple neuritis is more common in males than in females in all varieties. All ages are liable to be affected. The diphtheritic type is of course more commonly seen in children than in adults, and if these cases are included in a general list it would appear as if children were more subject to the disease than adults. In 154 miscellaneous cases, not diphtheritic, 16 were between the ages of sixteen and twenty years, 34 were between the ages of twenty and thirty years, 54 were between the ages of thirty and forty years, 35 were between the ages of forty and fifty years, 19 were between the ages of fifty and sixty years, and 10 were over sixty years of age.

Heredity appears to play little or no part in the causation of multiple neuritis, excepting in so far as the general tendencies to gout, rheumatism, diabetes, carcinoma, arterial sclerosis, and tuberculosis may be said to be hereditary.

It seems probable that in many cases a number of different causes combine to start the neuritis. Thus in a person who has used alcohol for some years the disease may not develop until some acute illness, like an attack of the grippe, or of rheumatism, or an exposure to cold occurs, when a typical alcoholic neuritis may manifest itself. The particular elements of causation will be more fully discussed in connection with the various types of the affection.

Symptoms. A general statement of the symptoms occurring in any or all forms of multiple neuritis will be made, and then the special combination of these symptoms, together with the common course of the disease in the different classes of cases, will be presented. Some authors have attempted a symptomatic classification of the forms of neuritis, and distinguish between sensory, motor, and ataxic types of the disease.[1] As many cases, however, present a combination of these symptoms such a division does not appear to me to be scientifically justifiable, however convenient from the clinical standpoint.

The sensory symptoms of multiple neuritis are the earliest to appear and the last to pass away. In the majority of the cases on record, from whatever cause, numbness, tingling, or formication ushers in the disease. These forms of paræsthesiæ begin in the feet and hands and extend to the knees and elbows. They may be associated with burning, stretching, boring, or tearing sensations which distress the patient especially during the onset, and such sensations usually increase as the affection reaches its height. Their subsidence, as the case goes on, may be regarded as a favorable symptom, but they are among the last evidences of the disease to disappear. Pain is usually present as well as paræsthesiæ. It is sharp in character and is usually intermittent. At times it may be lancinating and so severe as to necessitate the use of morphine. It is fully as distressing as in cases of locomotor ataxia. Tenderness in the nerves and muscles is a constant symptom. It may be so extreme that the limbs cannot be moved or handled, and thus it may interfere with the application of electricity and massage. When the tenderness and pain are referred to the joints, as not infrequently occurs in the early stage of the disease and in the spontaneous and infectious cases, the case may be mistaken for one of acute articular rheumatism; and if the joints are swollen or the limbs œdematous the difficulty of diagnosis is greatly increased.

In addition to these subjective feelings some demonstrable disturbance of the various sensations is usually present. Hyperæsthesia to touch and also to electricity is not infrequently observed during the first two weeks. It is usually followed by some anæsthesia, although this rarely becomes complete. In some cases the loss of tactile sense is quite evident from the onset, either limited to the cutaneous distribution of some special nerve, in which case oddly-shaped areas of insensibility will be found, as in lepra, or, as is most often the case, about uniformly distributed over the distal parts of the extremities and fading off into normal conditions higher up upon the limb. When the anæsthesia is at its height the patient has difficulty in locating a touch upon the hands and feet, even though he feels it. The distribution of the anæsthesia when it is fully developed corresponds to the parts of the extremities covered by gloves and stockings, and hence has been named the glove-shaped and stocking-shaped area of anæsthesia. (Fig. 27.) This distribution of anæsthesia, contrasting sharply, as it does, with that due to spinal-cord disease, and being uniformly symmetrical upon the two limbs, is quite diagnostic of multiple neuritis. The transmission of pain and temperature sensations is sometimes delayed, but these impressions are usually felt quite acutely. The sense of pressure has been tested in only a few cases, and in those it was decidedly impaired.

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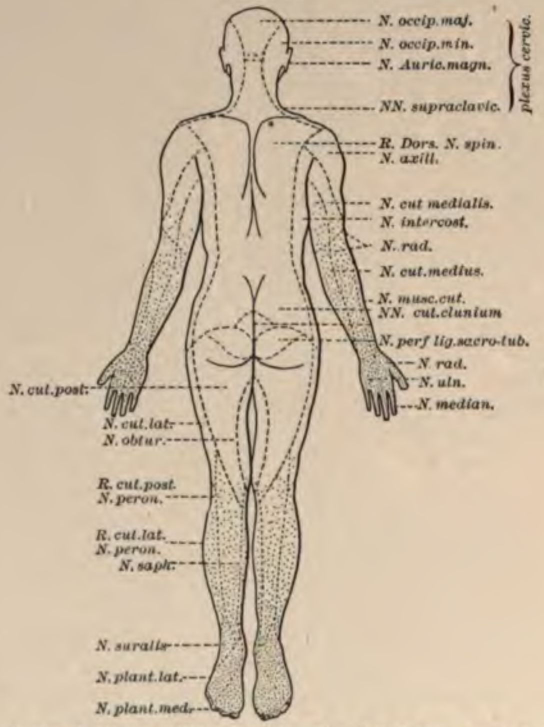
[1] Judson Bury. Clifford Allbutt's System of Medicine, vol. vi.

FIG. 27.



The distribution of anæsthesia in multiple neuritis. The dotted areas are anæsthetic. These do not correspond with the distribution of the various nerves.

The muscular sense escapes any affection in some cases, but in others is the most profoundly disturbed of all the senses. When it is involved the inco-ordination and ataxia are well-marked symptoms, and some cases have been mistaken for locomotor ataxia because of the predominance of the disturbance of muscular sense.

The ataxia is very pronounced in diphtheritic cases, in cases due to arsenical poisoning, and in some cases due to alcoholic poisoning. The French and Germans distinguish a class of cases of neuritis which they term *neurotabes peripherique* or pseudo-tabes, because of the close resemblance to locomotor ataxia.

These sensory symptoms are usually limited to the forearms and hands and to the legs and feet. In no case have they involved the entire extremities or the trunk, and but one case of facial tingling with anæsthesia has been recorded.[1] The skin reflexes are usually preserved and are occasionally exaggerated.

The special senses are rarely involved in multiple neuritis. It is true that optic neuritis has occurred in a few cases, especially in cases due to alcoholic poisoning, and in some cases hearing as well as sight has been affected. These cases prove that no nerve can be said to be exempt from implication in this disease, but the liability to affection seems to be slight in the case of the nerves of special sense. In alcoholic cases toxic amblyopia has been found.

The motor symptoms are as marked and as important as the sensory. Paralysis, beginning as simple weakness, with a feeling of fatigue on any exertion, gradually increases in severity until at the height of the disease it becomes complete. It usually comes on rapidly, so that within two weeks the patient is helpless; but it may be less sudden and not deprive him of the power of walking and of using his hands for two or three months. It may be arrested in the slighter cases and not go beyond a condition of general feebleness in the extremities. In a few cases a very acute onset is recorded, all the symptoms developing within three or four hours.

The distribution of the paralysis is not uniform at the outset. It may begin in the muscles of the legs and then involve those of the forearms; it may commence in all four extremities at once. It is always more severe in the muscles which move the joints of the feet and hands and the ankles and wrists. It rarely invades those which move the knees and elbows. The extensors of the wrists and fingers and the peronei and anterior tibial muscles in the legs are the muscles first affected, and it is usual for the paralysis to become more complete and to last longer in these muscles than in the flexors of the wrist or in the posterior tibial muscles of the legs. A marked tremor in the hands is not at all uncommon in the early stage of paralysis, especially in the alcoholic form, and usually the weakness is attended by slight inco-ordination and unsteadiness of gait before the patient is finally incapacitated from walking. When the disease is fully developed all the muscles below the knees and elbows are much weakened or totally paralyzed. Occasionally those of the thighs and arms are involved also, and the muscles of the trunk and those of respiration may become affected, and then the patient usually dies.

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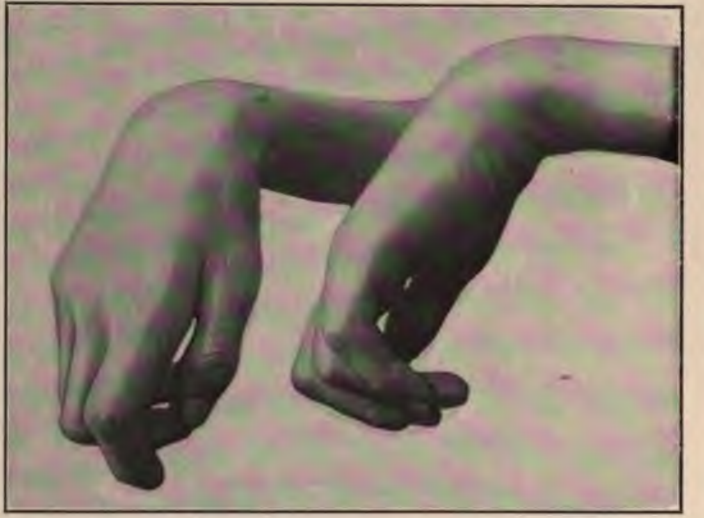
[1] Lowenfeld. Neurol. Centralbl., 1885, No. 7.

In two cases which eventually recovered I have seen a total paralysis of the diaphragm lasting several days. In both these cases the patient's respiration ceased as soon as he fell asleep, and the exhaustion from the long wakefulness and the voluntary efforts of breathing was very great.

In some cases of multiple neuritis the cranial motor nerves become involved, those of the eye and of the face being most liable to invasion. It is only in fatal cases that the action of deglutition has been affected, and when the pneumogastric is invaded and the heart becomes rapid and irregular the prognosis is always grave, though not absolutely bad.

FIG. 28.

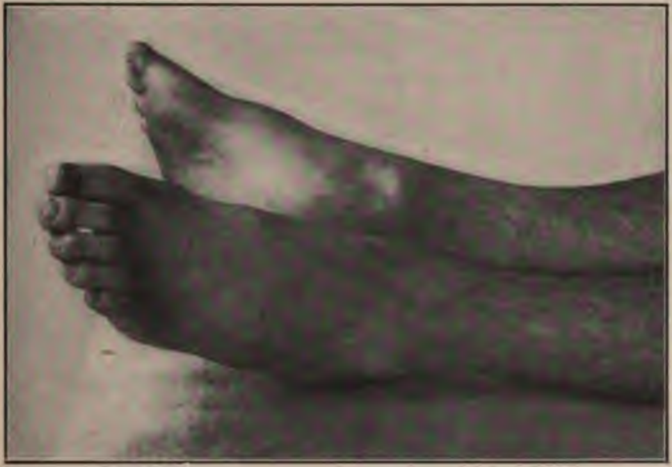


Dropped wrists in multiple neuritis.

The paralyzed muscles are relaxed, flabby, and atrophied; they may or may not lose their mechanical irritability, but their normal tone is always lost, and hence their tendon reflexes are abolished. The loss of knee-jerk is an early symptom in the disease, though some cases have been observed in which it has been preserved. These were cases in which the neuritis did not advance up the legs sufficiently high to affect the nerves above the knee. The knee-jerk always disappears early in diphtheritic cases.

To the electric current their excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost, and then the muscles react to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and this fact is quite pathognomonic of neuritis, for in anterior poliomyelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion. The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present; but in some cases the normal condition is found, and the negative pole produces stronger contractions than the positive pole. If the muscles which are not paralyzed be tested the same electrical changes may often be discovered in them. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are, therefore, important symptoms of multiple neuritis, and, as the disease goes on to recovery, a gradual increase in the galvanic irritability occurs, a fact which is often of much aid in prognosis if careful measurements of the strength of current used be made by the galvanometer

FIG. 29.

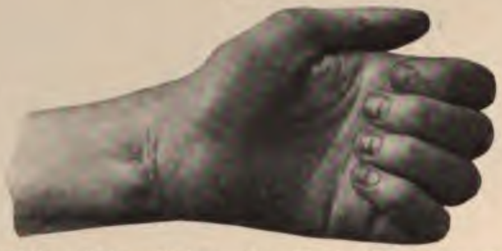


Dropped feet in multiple neuritis.

As a result and accompaniment of the paralysis abnormal positions are assumed by the limbs. The dropped wrist and dropped foot are quite characteristic of multiple neuritis. (Figs. 28 and 29.) They are in part due to the action of gravitation and in part to the unopposed action of muscles which are not very weak. But other deformities may be present. In a few cases there have been extreme contractures of all the extremities in flexed position. When the legs are thus flexed the posture is at first voluntarily assumed to relieve the pain, and later the flexor muscles become permanently contractured and shortened. These contractures may be exceedingly painful. In the severe cases a typical claw-hand and talipes equinus are seen. These deformities usually subside as the power returns, or, if they do not, they can be corrected by proper manipulation and by apparatus. In a few cases it has been necessary to resort to tenotomy, but a permanent deformity has not been recorded.

The vasomotor and trophic symptoms are less constant than those already described. In some cases marked œdema has been an early and a permanent symptom. This may develop in the feet and hands, or may appear about the joints. It is usually temporary. The circulation is not impaired to any greater degree than is customary in a limb whose muscles are inactive, and coldness and cyanosis are rarely sufficient to attract attention. Sometimes profuse perspiration is a noticeable symptom, being limited to the paralyzed parts. This is often seen in alcoholic cases. It may be offensive, and by its evaporation always causes a complaint of coldness. In other cases glossy skin makes its appearance early and remains until the regeneration of the nerves is complete. Its disappearance in one of my own cases was the first sign of recovery in the lower extremities. A change in the appearance and growth of the nails is very common, and a high ridge across the nail becomes evident as recovery begins, indicating the difference between the normal and abnormal nail formations. This is shown in Fig. 30. Bed-sores never appear. Other forms of trophic disturbance are rarely met with in multiple neuritis, and this is quite remarkable in view of the fact that it has been the tendency of late to refer such trophic affections as ulcerations, bed-sores, gangrene, pemphigus, and various eruptions to lesions of the nerves. It is true that inflammations of the joints resembling those appearing in acute rheumatism sometimes occur at the onset of neuritis; but as they disappear quickly while other symptoms remain, it is improbable that they are to be traced to the changes in the nerves. They may be due to the infectious agent or to the same

FIG. 30.



Ridged appearance of the nails in multiple neuritis. The hand is also atrophied, and the thenar eminence flat. The thumb cannot be apposed to the fingers.

obscure cause which sets up the neuritis, or they may be evidence of an attack of acute articular rheumatism, which is in turn followed by neuritis, but they cannot be described as trophic symptoms of the disease, otherwise they would be more constant in their occurrence and more permanent in their duration.

A negative symptom of great importance is the absence of any interference with the automatic action of the bladder or rectum. Retention or incontinence of urine does not occur, and impaction within or a relaxation of the rectum is not observed in the course of the disease. Constipation may ensue upon the long-continued rest in bed that is necessitated by the paralysis; but it is by no means as obstinate or as difficult to relieve as it is in spinal-cord affections.

Another negative symptom of some importance is the absence of pain or of anæsthesia upon the trunk. In the various forms of spinal-cord disease which are likely to be confounded with neuritis, zones of anæsthesia upon the body are not at all uncommon. It is well known that in locomotor ataxia bands of anæsthesia about the trunk and in the axillæ are almost constant signs of the affection. It is well to recognize the absence of this symptom in those cases of multiple neuritis which simulate locomotor ataxia.

There are certain mental symptoms which develop during the course of alcoholic multiple neuritis, but as they do not appear in the other types they will be discussed in connection with the toxic cases.

Course. The course of the disease in multiple neuritis varies so exceedingly that no general statement can be made. In the next chapters in which the various forms of the affection are considered the course of the case in each form will be described. General constitutional disturbances, however, are not infrequent in the course of multiple neuritis, and may be mentioned here. The onset is often sudden and accompanied by a marked febrile movement with chill and temperature of 103° or 104° F. The fever may persist for several days, but usually subsides spontaneously and does not recur. In a few cases there has been a constant elevation of temperature of about one degree, persisting for several weeks. The general symptoms accompanying fever, viz., malaise, loss of appetite, nausea, disturbances of digestion, constipation, flatulence, occasional diarrhœa, febrile condition of the urine, general pains all over the body, headache, and discomfort usually pass off as this subsides. An enlargement of the spleen and a condition of leucocytosis has been discovered by examination during the onset in some cases, especially in those following an infectious disease and in cases that are not due to poisoning. These facts have led to the theory that neuritis may be, under some circumstances, a primary infectious disease.

Increased rapidity of the pulse is very common in all forms of neuritis, and may persist during the entire course of the disease, the pulse ranging from 80 to 100. In some cases it becomes exceedingly rapid, reaching 140 or 160, being feeble and small. In these cases the neuritis has extended to the pneumogastric nerve. If this condition persists for several days œdema of the extremities and finally of the lungs may result, and heart failure may be the primary cause of death.

The duration of a condition of multiple neuritis is so different in different types of cases that no general statement is warranted. In the diphtheritic cases recovery is quite rapid, and I have seen patients who had been completely paralyzed quite well within two months. In cases of alcoholic neuritis of a mild type, where no actual paralysis developed, recovery was usually complete in six months. In the severer cases, where drop-foot and drop-wrist had developed, at least a year, and in several cases two years elapsed before their health and power were restored. Arsenic and lead cases are also slow in their progress, from eight to twelve months being the usual duration. The duration is in exact ratio with the degree of degeneration present at the maximum of the symptoms.

Diagnosis. While the individual symptoms occurring in the course of multiple neuritis are not different in character from those found in spinal-cord diseases, the diagnosis can usually be reached with very little difficulty when their combination, the causation, and the course of the case under examination are considered. There are three combinations of symptoms in neuritis which resemble very closely, respectively, anterior poliomyelitis, locomotor ataxia, and diffuse myelitis, and to these attention must be directed.

Atrophic paralysis, with reaction of degeneration and loss of reflexes, is common to anterior poliomyelitis and some cases of multiple neuritis. Poliomyelitis attacks healthy children, and no cause, as a rule, can be found. In neuritis it is often possible to ascertain some previous condition of ill health or some infectious disease or constitutional state which has produced the affection. In neuritis, a more gradual onset, preceded and attended by numbness and pain, tenderness in the course of the nerves, tenderness in the muscles, and the persistence of sensory symptoms after the invasion will remove all doubt regarding the diagnosis. When these symptoms are not clearly marked the distribution of the paralysis in symmetrically situated muscles, especially if these muscles are supplied by single nerves, and the further extension to muscles in other nerve domains, rather than the affection simultaneously of muscles which are grouped physiologically (*i. e.*, act together to perform one function) will point to neuritis. In neuritis the paralysis advances more or less gradually, while in acute poliomyelitis there is, after the onset, a subsidence of the paralysis in some of the muscles first involved. In neuritis the cranial nerves are not infrequently affected. This does not occur in poliomyelitis. Ataxia, which is a common symptom in neuritis, never appears in infantile paralysis. Cramps in the muscles are complained of in neuritis, but not in poliomyelitis. In the latter fibrillary twitchings occur in the muscles which are paralyzed, but never in the former. Glossy skin never appears in poliomyelitis. Lastly, as the case goes on, a gradual complete recovery will be far more frequent if it was originally a case of multiple neuritis. Gowers has described a number of cases in which, he believes, the two diseases have occurred together, probably being produced by the same cause. Under these circumstances a mingling of the symptoms is to be expected, and no sharp differentiation can be made.

Ataxia, loss of knee-jerk, pain, and sensory disturbances, including a loss of muscular sense, Romberg's symptom, that is, swaying when standing with closed eyes, and optic neuritis, are common to locomotor ataxia and to multiple neuritis. This form of neuritis has been called acute polyneuritic ataxia or neurotabes peripherica by Dejerine,[1] who was the first to point out clearly its resemblance to locomotor ataxia. In neuritis the relatively rapid onset of the ataxia, which follows closely upon the sensory symptoms; the prominence of numbness and anæsthesia, rather than of lightning pains; the extreme degree of the anæsthesia and analgesia, the tenderness of muscles and nerves, the usual occurrence of some degree of actual paresis, with atrophy and reaction of degeneration, and the absence of bladder and sexual symptoms, will point inevitably to the correct diagnosis. Furthermore, the ataxic form of neuritis only occurs after poisoning with alcohol or arsenic, or as a sequel of diphtheria, and the establishment of the causation will aid the diagnosis. Here again, the course of the case toward recovery and the return of the knee-jerk will decide in favor of neuritis if the diagnosis has not been reached in an early stage.

There are very few symptoms of diffuse myelitis which are not found in cases of neuritis, but cases of diffuse myelitis of the type described by Duchenne (*paralysie générale spinale subäigue ascendante*) are very rare, and, indeed, it has been affirmed by Leyden that the cases described by Duchenne under this name were really cases of multiple neuritis. A differential diagnosis between general myelitis and neuritis is made by a consideration of the following points: In neuritis affections of the functions of micturition and defecation do not occur. Girdle sensation is very rarely mentioned as a symptom. Bed-sores and cystitis have not been observed. In neuritis the advance of the paralysis is not like that in myelitis, namely, a gradual advance from legs to thighs, and thighs to trunk, and trunk to arms and neck; but, as already stated, the paralysis begins in the legs and forearms simultaneously, and does not usually extend to the thighs and arms and very seldom invades the trunk. If the muscles of the abdomen and the muscles of respiration are involved in neuritis it is only in the rapidly fatal toxic cases, and even in these cases motions of the shoulders and hips are preserved until the end. In the type of myelitis described by Duchenne there are few sensory symptoms, whereas these are prominent in neuritis. If in myelitis there are sensory disturbances and anæsthesia the areas are not glove-shaped and stocking-shaped and there is a well-marked line of demarcation around the trunk. This is particularly evident in transverse myelitis. In neuritis, however, the anæsthesia is chiefly observed in the hands and feet, in the forearms and legs, but rarely reaches as high as the arms or thighs. In neuritis there is usually tenderness in the nerves and in the muscles of the extremities which is not present in myelitis. In myelitis there is usually tenderness to pressure and sensitiveness to heat along the spine, a symptom not present in neuritis. In the older text-books multiple neuritis is not mentioned, and cases of it were described as cases of spinal-cord disease; but since the knowledge of neuritis has become general the diagnosis of myelitis is more and more rarely made, and now that disease is regarded as an unusual one.

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[1] Arch. phys. norm. et path., 1884, p. 231.

Prognosis. The prognosis in multiple neuritis is good, provided the exciting cause can be removed. The only patients who form an exception to the rule are those whose constitutions are much impaired by excesses or by other diseases; those who have so far indulged in alcohol or are so completely soaked with arsenic or lead as to be unable to throw off the poison, and those in whom the disease begins with great suddenness, advances rapidly, and involves the phrenic and pneumogastric nerves. These cases die either of respiratory paralysis or of some complication. When a case has reached the stationary period the prognosis is generally favorable, and if the encouraging signs of recovery already mentioned begin to appear a cure may be promised. The possibility of the complication of myelitis must not, however, be overlooked, and if it occurs the prognosis becomes at once unfavorable. Even in serious cases of alcoholism, with gastro-intestinal and cerebral symptoms, if the acute stage be safely passed and all alcohol be removed from the patient's diet, recovery from very extensive paralysis will occur. The cases of diphtheritic paralysis and ataxia usually recover without treatment.

Treatment. The treatment of multiple neuritis requires patience. As we have already seen, the majority of the patients recover, and it is probable that, if the cause of the affection were removed and the patients placed in favorable circumstances, expectant treatment would alone be sufficient. It is, however, not advisable to let therapeutics play a passive part. The course of the disease can be altered and its duration much shortened by active interference. In the stage of invasion the free use of salol, salophen, salicin, salicylic acid, or the salicylate of soda seems to have important results. These remedies cannot be said to act as promptly as in cases of acute articular rheumatism, but the consensus of opinion is that their effect in multiple neuritis is very marked. They should be given, as in acute rheumatic fever, in large doses until noticeable effects are obtained. They should be combined with the bromide of potassium or sodium, partly because these drugs counteract unfavorable symptoms produced by the salicin compounds and partly because in the hyperæsthetic irritable condition attendant upon the invasion of the disease they are indicated. This condition may require stronger sedatives and not infrequently morphine must be employed to give relief from the excruciating pains. The pains are often relieved by hot or cold applications to the limbs; but as the muscles are often exceedingly tender, ordinary applications cannot be made. It is then advisable to use evaporating lotions, preferably those containing chloroform, which may be soaked into light cambric or gauze and gently placed upon the limbs, which lie upon the softest pillows or which may be more comfortable if the patient is put upon a water-bed. Applications of a 5 per cent. solution of carbolic acid and of extract of witch-hazel have also been of use. If cool applications prove intolerable heat may be employed. The limbs may be enveloped with cotton and covered with oiled silk, a light bandage keeping these in place, or they may be frequently bathed in hot water and hot bottles placed against them, some soft substance intervening. One of my patients found great relief from the paræsthesia by cold douches, while another preferred the use of hot water. It is best to let the patient decide, as long as the application has to be made for the relief of pain. Gentle friction with oil of cocoanut or cocoa-butter often affords comfort. In the chronic stage, as we shall see presently, heat is to be preferred to cold. Cases which are distinctly syphilitic, if such occur, should be treated from the outset with inunctions of mercury and large doses of iodide of potash. I believe that both these drugs should be employed together, even in the tertiary stage of syphilis, and it is my experience that all syphilitic nervous lesions, whether central or peripheral, yield more promptly to their combined use than to the employment of either alone. Malarial cases must be treated with quinine or Warburg's tincture. In non-malarial cases quinine has proved of no avail.

In cases which are due to poisoning of any kind the first indication is to eliminate the toxic agent from the system and the second to prevent any further ingestion of the poison. Iodide of potash in small doses largely diluted aids in the elimination. The second indication is easily fulfilled when arsenic or lead are the toxic agents, but when the case is due to chronic alcoholism special precautions are needed. Alcoholic cases require from the outset special treatment. The condition at the time of the onset of the paralysis may be one verging upon delirium tremens. If all alcohol is suddenly removed, without due care to supply some other heart stimulant and to secure the perfect nutrition of the patient, serious collapse may ensue. The first necessity is, therefore to take care of the general condition of the patient. If this will admit of the immediate withdrawal of all alcoholic stimulation it should be done; if not, the alcoholic beverage must be immediately reduced in quantity, and as soon as possible wholly cut off. The use of milk diet, or kumyss, or peptonized milk, or, if necessary, rectal alimentation, will be followed by a gradual recovery of the power of assimilation, and as soon as the patient ceases to lose weight all alcohol may in any case be safely stopped; its elimination by the intestines and kidneys may be hastened by appropriate means, and cerebral symptoms, if they arise, may be treated as in other cases of alcoholic intoxication. But it is in the chronic stage, when the patient is gradually recovering, that the vigilance of the physician is called into play to prevent a renewal of the poisoning. It is amazing that patients who know perfectly the injurious effect of alcohol upon them should insist upon getting it; but it is done. And when these patients are surrounded, as is often the case, by sympathizing friends or servile domestics or unscrupulous nurses who do not appreciate the importance of total abstinence for the patient, they often succeed in baffling all attempts to deprive them of the favorite drink. It is only when they are watched constantly by persons who can be implicitly trusted and who have sufficient authority to cut off all surreptitious supplies, that the physician can feel sure that his commands are obeyed; and this precaution is by no means needless, even when it is probable that family servants are trustworthy, for the continued pleading and remonstrance of the patients may corrupt the best of attendants, especially if accompanied by threats of discharge at a future day. It is, therefore, necessary to place these patients under the surveillance of trained nurses from the start or to remove them to an institution where they will be under control.

In the chronic stage the drugs which are of greatest service are strychnine and arsenic. Strychnine may be given in doses of $\frac{1}{60}$ to $\frac{1}{40}$ gr. three times daily, and it is well to combine it with phosphoric acid and the syrup of the hypophosphites, or with the glycerophosphates of lime and soda. Arsenious acid may be used in tablets or pills containing $\frac{1}{50}$ to $\frac{1}{30}$ gr. three times daily, or in Fowler's solution, three to five drops, three times daily. The use of iron with these two drugs will be indicated in the majority of cases where there is attendant anæmia. In alcoholic cases both arsenic and strychnine may increase the mental irritability, but should be continued unless this becomes too great. I have seen benefit from both of these drugs, and think it well to employ them alternately, using each for about two weeks at a time. The glycero-phosphate of soda dissolved in water is of much service in the chronic stage. The dose is 15 grains three times daily.

The remedies used in the chronic stage have two objects: one is to increase the rate of repair in the nerves, the other is to keep the nutrition of the muscles as good as possible. While the drugs mentioned probably meet the first indication, there are other remedies which meet both. These are massage, warm baths, and electricity. The proper manipulation of the limb increases the circulation in it. The increase of circulation brings fresh supplies of material to the nerve which is undergoing repair; it also aids the nutrition of the muscle, which would otherwise be decidedly affected by the sluggish flow of venous blood, due to the lack of functional activity. As soon, therefore, as the active progress of the disease is checked and the muscular tenderness has sufficiently subsided to allow the limbs to be rubbed, this remedy should be employed daily.

Allusion has already been made to baths and douches in the early stage, for the purpose of quieting sensory symptoms. In the chronic stage the object is a different one. Like massage, warm baths, packs, and douches stimulate the circulation and aid the nutrition and reparative processes in progress. Hence they are to be used daily; and if the warm bath or pack be given at night it will secure not only a local action, but produce a general sedative effect, insuring quiet rest. In multiple neuritis, as well as in many other nervous affections not due to anæmia, a warm bath or pack at night or a warm douche to the neck and spine, is far preferable to and more efficacious than the majority of hypnotic drugs in causing a good night's sleep.

The last agent to be mentioned is electricity. It is, however, of some importance, but it is necessary to know the object sought in its application. There is first the object of increasing the progress of nerve regeneration. This is said to be attained by the application of a constant galvanic current to the degenerated nerve, passing the current through the nerve in either direction, or in both alternately. A mild current should be employed, its strength being measured by a galvanometer. The strength of the current will depend (*a*) on the size of the sponges placed upon the skin, (*b*) on the pressure upon the sponges, (*c*) on the resistance of the skin, and (*d*) on the number of cells of the battery used.

If the sponges are two inches in diameter, six milliampères are enough, if they are three by five inches in measurement, twenty milliampères should not be exceeded. If no galvanometer is used the strength of the current employed is uncertain; but it is to be remembered that the current grows stronger the longer it passes, since the skin resistance is gradually overcome; and, therefore, if the strength is measured in cells, the number of cells used should be decreased gradually during the application. As so few practitioners use a galvanometer, it may be well to state that with large sponges—*i. e.*, three by five inches—wet with warm water, a freshly filled bichromate of potash battery will give nearly one milliampère of strength for every cell used during the first three minutes, provided the sponges be put on any part of the body except the soles of the feet or the palms of the hands, and pressed firmly upon the skin. After the first three minutes the body resistance decreases, so that when twenty cells are used to start with, one should be cut off every half-minute until the number is reduced one-third. The duration of the application should be about ten minutes to each limb. During this time the distal sponge should be passed over various parts, so as to include all the nerve branches in the current; the central sponge should be put over the nerve trunk high up on the limb. The current should be begun and stopped gradually, and never suddenly broken. Applications may be made daily.

The second object to be obtained is to re-establish the conduction of impulses in the regenerated nerve. This is secured by the method just described. It may be attained by the use of faradism, the interrupted secondary current being sent along the nerves by placing one pole over the nerve trunk and passing the other over the skin of the limb. The strength used should be just sufficient to be felt distinctly through the palms of the operator's hands. The third object sought is to maintain the nutrition and function of the muscles by exercising them, and thus to preserve their normal irritability. This cannot be done by a faradic current as long as they do not contract to it. The galvanic current must, therefore, be employed. But now it is not a steady current which is needed, for this does not cause any motion. It is only when the steady current is suddenly broken and renewed that the contraction occurs. Hence, place one pole over the trunk of the nerve and the other upon the muscle, and with an interrupting electrode make and break the current at the pole which is on the muscle. The pole which produces a contraction with the least current possible is the one to be applied to the muscle. This is in the reaction of degeneration, the positive; in normal conditions, the negative pole. Each muscle should be exercised for three or four minutes every other day. When electrical treatment is thus employed very marked improvement is observed, which can be measured accurately if a galvanometer is used, since every week will show a change of the strength of current needed to produce muscular contractions toward the normal.

The treatment must be kept up, in the chronic stage, until recovery is complete.

If contractures have occurred in the paralyzed limbs, persistent massage may overcome them. If it does not, they are to be treated on general surgical principles. When the posterior tibial muscles are contractured, the patients cannot stand because the heels cannot be placed on the floor. In this condition shoes with very high rubber heels, and either corset-like ankles or braces to support the ankle, may be fitted, and thus the patient may be enabled to walk with a little help long before he can stand in his bare feet. Sometimes division of the Achilles tendon is necessary to restore the power of walking.

CHAPTER VI.

THE TOXIC FORMS OF MULTIPLE NEURITIS.

Alcoholic Neuritis. Multiple Neuritis from Poisoning by Illuminating Gas. Multiple Neuritis from Poisoning by Sulphide of Copper. Multiple Neuritis from Poisoning by the Coal-tar Products. Arsenical Neuritis. Multiple Neuritis Due to Poisoning by Lead. Multiple Neuritis Due to Poisoning by Phosphorus, Mercury, Silver, and Copper.

ALCOHOLIC NEURITIS.

Historical. Alcoholic neuritis is the most common of all the toxic forms of multiple neuritis. The credit of the discovery of alcoholic paralysis must be awarded to James Jackson, of Boston, who, in 1822, gave a most accurate description of the disease and ascribed it to the use of ardent spirits.[1] Magnus Huss, in 1852, published an extensive work on chronic alcoholism, in which he gave a very complete account of the nervous symptoms produced by the abuse of liquor, dividing the cases into paralytic, anæsthetic, convulsive, epileptic, and hyperæsthetic forms. But he ascribed all the symptoms to lesions of the central nervous system. In 1855 Duchenne de Boulogne, in his epoch-making book, *Electrisation Localisée,* recorded many cases which we now recognize as cases of neuritis; but he, too, supposed them to be of spinal origin. Lancereaux added further to the knowledge of alcoholic paralysis in an article on alcoholism in the *Dictionnaire Encyclopœdique des Sciences Médicales* in 1864, and in the same year Dumesnil[2] published the first case in which a lesion was found in the peripheral nerves. But ten years then elapsed before his observations were confirmed by Eichhorst,[3] and it required further evidence furnished by Joffroy,[4] by Leyden,[5] and by others to establish the relation between alcoholic paralysis and neuritis. It is to Lancereaux,[6] of Paris, to Moeli,[7] of Berlin, to Dreschfeld,[8] of Manchester, England, to Henry Hun,[9] of Albany, N. Y., and to Bernhardt,[10] of Berlin, that we owe our knowledge of the pathology and symptomatology of alcoholic multiple neuritis, though other and subsequent writers have added valuable facts and have made the picture of the condition complete. James Ross,[1] Brissaud,[2] Pal,[3] and Gowers[4] may be mentioned as having made interesting additions to the general knowledge of the affection, and as having clearly presented its differential diagnosis from other diseases. The most complete monograph on the subject is by Remak and Flatau in Nothnagel's *System of Medicine*, 1900.

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[1] New England Journal of Medicine and Surgery, 1822, vol. xi. p. 351, "On a Peculiar Disease Resulting from the Use of Ardent Spirits."

[2] Gaz. Heb. de Paris, 1864, p. 203, and 1866, No. 4.

[3] Virchow's Archiv, 1876, Bd. 69, p. 205.

[4] Arch. de phys. norm. et path., 1879, p. 172.

[5] Charité Annalen, 1880, Zeitschr. f. klin. Med., 1880.

[6] De la Paralysie Alcoolique, Gaz. Heb. de Paris, 1881, p. 120.

[7] Brain, Nos. 26 and 32.

[8] Moeli. Charité Annalen, 1884.

[9] American Journal of the Medical Sciences, April, 1885.

[10] Zeitsch. f. klin. Med., 1886.

Pathology. All forms of neuritis have been found in alcoholic cases. In the majority of cases a parenchymatous neuritis with simple degeneration and atrophy is the only lesion in the nerves. (Plate VIII.) In other cases a diffuse inflammation is present, with an increase of connective tissue in the endoneurium and perineurium, and a marked congestion of the bloodvessels, as well as a degeneration of the nerve fibres. All the characteristic changes already described as occurring in local neuritis are present in alcoholic cases. But it is not possible to distinguish clinically between cases in which the lesion is a simple degeneration, and cases in which there is a diffuse inflammation. The lesion is more marked in the finer branches of the nerves, in the skin and muscles, and becomes less intense the higher up the nerve is examined. In the plexuses no change may be found, when at the same time few normal fibres can be discovered below the wrists and ankles. Certain nerves seem particularly susceptible to the influence of alcohol. The degeneration begins in the peronei, the tibials, then in the radial, median, and ulnar nerves, and is more intense in these than elsewhere. (See Plate IX.) The sciatic and crural and the musculocutaneous and circumflex are but slightly affected. Lesions have been found in the phrenic, in the pneumogastric, and in the optic nerves. Normal fibres are found here and there in the nerves which are severely affected. A thickening of the bloodvessels in the nerves has been discovered in some cases, with a small-cell infiltration of the walls, and occasional rupture, with capillary hemorrhages.

Recent observation establishes the fact that while the principal changes in alcoholic paralysis are found in the peripheral branches of the nerves in all cases, yet in some cases changes in the cells of the central nervous system are present. These have been discovered in the cells of the anterior horns of the spinal cord and in the posterior spinal ganglia.[5] It is evident, therefore, that while the poison circulating in the blood produces a destructive process in the delicate filaments of nerve fibres in the periphery, yet the central nervous system is by no means free from its action. The cells of the spinal cord are less affected by the poisoning than the more highly organized and developed cells of the brain. The changes in the cord cells

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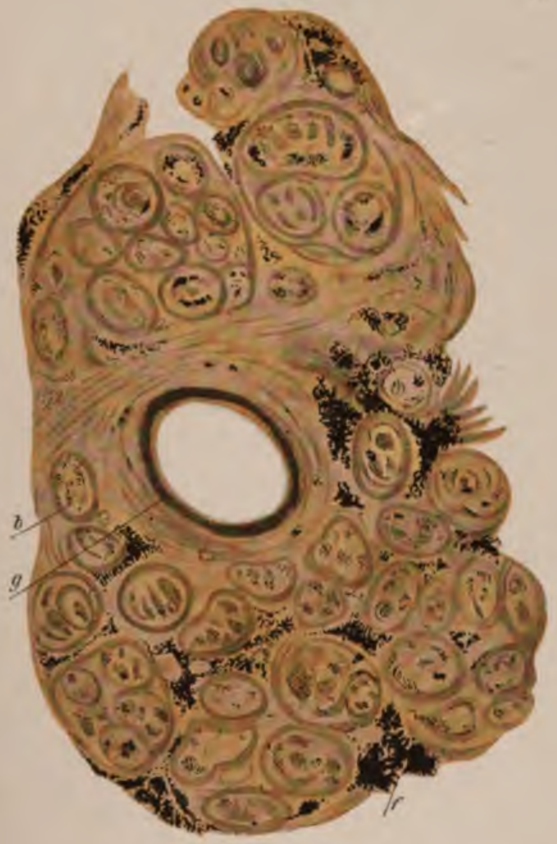
[1] James Ross and Judson Bury. "On Peripheral Neuritis," 1889.
[2] Brissaud. Les Paralysies Toxiques, Paris, 1890.
[3] Pal. "Ueber Neuritis," Berlin, 1892.
[4] Gowers. Text-book of Nervous Diseases, 1892, 2d ed.
[5] Larkin and Jelliffe. N. Y. Med. Record, July 8, 1899.

PLATE VIII.

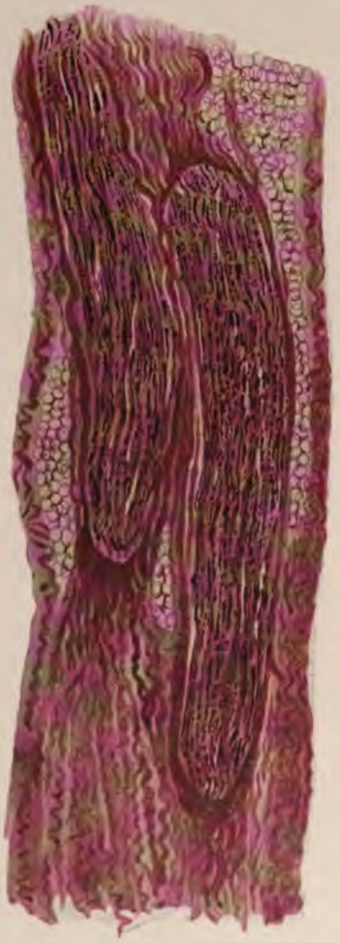


Cross-section of the Brachial Plexus in a Case of Neuritis.
(Marchi stain.)

The degenerated fibres in the different bundles of nerves (*b*) are stained black. In every bundle a number of such fibres are to be seen. *g.* artery ; *f.* fat.

(Flatau, Spec. Pathol. u. Therap., Nothnagel, Bd. xi , Taf. iii.)

PLATE IX.



Longitudinal Section of Tibial Nerve in a Case of Alcoholic Neuritis. (Osmic acid and carmine stain.)

The degenerated nerve fibres are stained black; the thickened interstitial connective tissue is stained red.

(Flatau, Spec. Pathol. u. Therap., Nothnagel, Bd. xi., Taf. vii.)

consist of a change in the Nissl granules, which become finely granular, and lose, finally, their power of absorbing anilin dye. This change is more marked about the nucleus and in the centre of the cell body which has a pale appearance. (See Plate I, G.) There is often a deposit of pigment in the cells. As these changes, however, have been found in all the cells of the cord, even in those connected with muscles which were not paralyzed, and as they resemble closely the changes known to occur after division of a nerve, they are not to be regarded as the cause of alcoholic paralysis. Heilbronner[1] has found an ascending degeneration in the posterior columns of the spinal cord in an advanced case of alcoholic neuritis, and is inclined to trace it to extensive degeneration in the posterior spinal neurones, but this is certainly an exceptional case.

Berkeley, Andriesen and Van Gieson have demonstrated a uniform pathological condition produced in the large cortical cells of the brain in chronic alcoholism, and these changes have been frequently found in cases of alcoholic neuritis. The dendrites of these cells lose the fine granules which cover them, and become smooth and bare. (See Fig. 4.) Then the dendrites become broken and disintegrated. The cells become swollen, vacuolated, degenerated, and finally atrophied, and their axones share in the destruction. So long as the lesion is limited to the shedding of the granules it is not irreparable, and regeneration may occur. In the cases of alcoholic neuritis which present psychical symptoms, there is every reason to suppose that these pathological changes are present in the brain, and the degree of impairment of mental capacity is directly proportionate to their intensity. A general atrophy of the muscular fibres is always present in the paralyzed muscle, with a loss of their striation, a fatty degeneration and a disintegration of the muscular substance, and an increase of the connective tissue. The muscular fibres show great irregularity of size, and present both parenchymatous and interstitial changes with the deposit of fat.

Etiology. Males are more commonly affected than females by alcoholic paralysis. Thus in 250 cases which I have collected or observed but 89 were in women. When, however, the much greater prevalence of drinking among men is considered, it becomes evident that the disease is relatively far more frequent among women than among men. And this fact has been emphasized by all writers. In men the poison seems more liable to manifest itself by acute cerebral symptoms than by those of disease of the peripheral nerves. Neuritis is especially frequent among those persons in the higher classes whose nervous organism is highly developed, and who lead a comparatively inactive life. It seems not at all improbable that sedentary habits predispose an alcoholic drinker to this disease, and hence active workers, male or female, though taking an equally large amount of liquor as the luxurious drinker, escape.

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[1] Heilbronner. Monatsschrift f. Psych. u. Neurologie, 1898, p. 246.

All alcoholic drinks are not equally prone to produce paralysis. It is the spirituous liquors—brandy, whiskey, gin, and rum, and the liqueurs, absinthe, vermouthe, etc.—which are particularly dangerous. But many cases are known to be due to excessive beer drinking. I see every year at the clinic cases in drivers of beer wagons who consume forty or fifty glasses of beer daily. I have even seen a case in a child of three years who had been given beer several times a day for some weeks. I have known cases in women to be due to taking cologne; and in Ireland, where ether is used as a drink, many cases are due to this cause. The fumes of alcohol are capable of causing the disease in a mild form. It is steady drinking of small amounts throughout the day, rather than excessive occasional indulgence, which is more likely to cause neuritis. The liability to neuritis from medicinal uses of alcohol should never be forgotten in those cases where it is used as a heart stimulant. I have seen many cases developing after typhoid fever and pneumonia where it seemed very probable that the actual cause was the free use of alcoholic stimulants given to support the heart, little food being given. All ages are liable to be affected, but the majority of cases are between thirty-five and fifty years of age.

Symptoms. It is only after alcohol has been consumed in large amounts and for a considerable length of time that neuritis develops. Its onset, though often apparently gradual, is usually sudden. For months the patient has suffered from chronic gastritis, insomnia, general neuralgic pains, or severe pains in the joints or limbs, and from alcoholic tremor and a certain feebleness in movement, when all at once his legs give way beneath him and after the sudden fall he finds himself unable to rise. Thus a patient of mine, after a year of such premonitory symptoms, was seized with paralysis quite unexpectedly when getting out of bed in the night. The onset in other cases is preceded by symptoms of disturbance in the heart and lungs. The patient has had palpitation and dyspnœa; has had swelling of the feet, undue exhaustion on exertion and possibly a bronchial cough; and when examined is found to have a feeble heart, dilated and flabby, with faint sounds or distinct mitral regurgitant murmur, and the signs of chronic pulmonary congestion. Then, after a period of unusual indulgence in stimulants, paralysis suddenly develops with all the sensory disturbances of a neuritis.

When the paralysis begins it soon becomes complete in the feet and legs to the knee, and may advance up to the thigh involving the extensors of the legs. It next attacks the hands and forearms, and while in all extremities it is greater in the extensors than in the flexors, in some cases both groups of muscles become entirely helpless. The paralyzed muscles are flabby, and soon become atrophied, they have no excitability to mechanical irritation, and the tendon reflexes are lost. They fail to react to a faradic current in the majority of cases, though occasionally a very strong current may produce a response. When galvanism is applied the reaction of degeneration is found to be present. It is often found that strong galvanic currents have to be used to produce any contraction at all. The paralysis of the muscles may advance rapidly in severe cases, involving the motor cranial nerves, the muscles of the trunk, and, lastly, the diaphragm, thus causing death. More frequently, however, it is arrested when only the distal parts of the extremities are involved, then, after a stationary period, which varies from a few weeks to several months, it gradually subsides until recovery is complete.

The position assumed by the paralyzed limbs has been thought to be almost characteristic. Dropped-wrist and dropped-foot appear early, due to the paralysis of the extensors, and further deformities of hands and feet follow. When the feet are dropped the first joint of the toes may be hyperextended though the second is flexed, but as time goes on this hyperextension gives place to flexion and then the entire foot is paralyzed and hangs down, the natural concavity of the sole being increased. The weight of the bedclothes may increase the dropping of the feet and flexion of the toes. Later the muscles of the sole and of the calf become permanently shortened and the sole can no longer be placed on the floor. When the patient is able to begin to stand, all the weight comes on the toes, which often turn under. Then the heel of the shoe has to be built up in order to form any support. The deformity of the hands is also different in different stages of the disease. At first there is dropped-wrist, the fingers hanging limp. Later, a claw-hand appears, the first phalanges being hyperextended; the second and third flexed. The thumb is also hyperextended, and the metacarpal bone being drawn backward but adducted, the thumb can no longer be apposed to the fingers. (See Fig. 30.) After a time the hyperextension gives way, the intrinsic muscles of the hand being paralyzed and the fingers are straight, adducted, and immovable, being fixed by contractured, atrophied muscles. In this state ankylosis of the smaller joints often develops, and the use of the hands is very slowly regained after painful massage and passive motions. I have seen bilateral facial paralysis of the peripheral type in a case of alcoholic paralysis; and several cases in which the ocular muscles were paralyzed. Irregular, rapid pulse, due to an affection of the pneumogastric, is a not uncommon occurrence in alcoholic cases, and is a dangerous symptom.

The patient suffers far more from the disturbance of sensation than from the paralysis. In the description of Jackson, the pains were graphically portrayed. They are the cause of terrible agony, are sufficient to produce insomnia, and wear seriously upon the endurance of the sufferer.

In addition to pain, hyperæsthesia, both of the skin and muscles, is always observed. It is usually quite extensive in the legs, though in cases of poisoning by absinthe it has been limited to the soles of the feet. The muscles, as well as the skin, are sensitive to handling and to pressure, and marked tenderness in the course of the nerves is constantly elicited by examination. In many cases soon after the onset, the patients cannot bear to be touched or moved, though perfectly unable to help themselves. Cramps in the calves or in the extensor surface of the thighs are often complained of and give rise to much distress.

Paræsthesiæ are always complained of. Numbness, tingling, and formication are frequent. In one of my patients the sensation was as if heavy bracelets were around the wrists, and as if very tight drawers were on the legs. At other times she felt as if the limbs were swollen, and as if the skin was about to burst. One patient described the sensation as being like iron stockings on the legs. Another insisted that there were needles under the skin of the hands. Such sensations may cease as the case increases in severity, and give place to a total lack of sensation in the parts. They return, however, with advancing recovery, and are among the last symptoms to disappear.

Abolition of tactile sense, and to some degree of muscular sense, is the rule after the paralysis has developed. Temperature sense and the perception of pain are never wholly lost, but may be delayed in transmission. Thus in multiple neuritis the phenomena of dissociated sensation may be found, but in the reverse sense to that observed in syringomyelia. The anæsthesia may be limited to irregular areas, and may be found in the cutaneous distribution of one nerve only, but is usually found over the entire distal part of the paralyzed limb. Usually the cutaneous reflexes are preserved.

The loss of muscular sense is, in some cases, so marked a symptom, and one of such early occurrence, that Dreschfeld distinguished a class of cases which he terms ataxic rather than paralytic. And this distinction is perfectly justifiable, for in many cases it is the incoordination which first attracts the attention of both the patient and the physician. It is this class of alcoholic cases which may be mistaken for locomotor ataxia, and which have been named by French writers *pseudo-tabes alcoolique*,[1] or neurotabes peripherica by the Germans. But ataxia is not exclusively limited to this class of cases. It may be present in some degree in cases of paralysis, and during recovery from paralysis the deficiency in co-ordinating power may become evident, and appear to retard the progress of the case. Nor are the cases of ataxia, on the other hand, free from paralysis.

The gait in neuritis is quite characteristic. By contrasting the walk of a true ataxic patient with that of an ataxic alcoholic patient, Westphal[2] and Charcot[3] have each pointed out several points of difference. The tabetic patient throws the foot forward with undue violence, the toe lifted high in the air, and brings first the heel down forcibly and then the entire foot. The alcoholic, however, has some weakness in the muscles of extension and cannot raise the toe.

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[1] Dejerine. Arch. de phys., 1884. See also Dreschfeld, Brain, Nos. 26 and 32.

[2] Westphal. Ueber eine bei chronischen Alcoholisten beobachtete Form von Gehstorungen, Charité Annalen, 1879.

[3] Charcot. Leçons, Le Progrès Médicale, 1886.

He, therefore, lifts the foot high in order to step over the hanging toe and not to trip on it, but the motion is made without undue force. He then throws the foot forward in order to throw the toes up and get them out of the way as he brings the foot down to the floor. The motion is awkward, and has an appearance of one stepping over high obstacles, but it is a voluntary attempt to remedy a deficient power —not the involuntary awkwardness of a man unable to manage strong muscles. There may be in both patients some tottering and swaying when standing with the eyes closed; this so-called Romberg symptom is common to both locomotor ataxia and alcoholic neuritis.

The vasomotor symptoms in alcoholic neuritis are very variable. Sometimes they precede the attack of paralysis for some months. There are irregularities of circulation in the extremities causing cold hands and feet, or burning red hands and feet, or undue sweating. When the paralysis is developed the color of the extremities is pale; they are cool and often wet with perspiration or occasionally livid and hot. If allowed to hang down both hands and feet get purple and swell. Ross saw one case in which Raynaud's disease developed. As the disease goes on, glossy skin appears and then the dermal tissues become atrophied and the skin seems too tight for the fingers and toes. It is often pigmented and scaly, and, when the tenderness is so great as to prevent washing, dark brown, scaly masses may collect on the fingers and hands and feet. The growth of the nails is always affected. They become ridged transversely, are thick and brittle, and too tender to be cut. As recovery begins new, thin, healthy nail appears above the thick ridge. (See Fig. 30.)

The special senses are occasionally affected in cases of alcoholic paralysis. Amblyopia has been observed, and also defective vision from central scotoma. The field of color vision is often contracted even when sight is preserved. There may develop a true optic neuritis, evident to the ophthalmoscope, and this may go on to optic nerve atrophy.[1] Inequality of the pupils is frequently seen, as is also a moderate contraction of the pupil. All these eye symptoms, occurring as they may in a case of the ataxic variety, make a differential diagnosis from locomotor ataxia difficult. The Argyll-Robertson pupil (which contracts in accommodation, but not to light) has not been seen in alcoholic cases, while it is an early symptom of tabes. One writer affirms that a condition of the pupil just the reverse of the Argyll-Robertson pupil is present in alcoholic neuritis, a pupil which reacts to light but fails to react in accommodation. I have been unable to confirm this statement.

One important feature of alcoholic paralysis remains to be noticed, viz., the mental symptoms. These are hardly ever wanting in severe cases. I called attention to these symptoms in 1887.[2] They have been most fully described by Korsakow[3] and Soukhanoff.[4] There is at first excitement rising to the degree of active delirium, with illusions and hallucinations of the various senses; there is insomnia, which soon exhausts the patient if it is not remedied; there is a loss of memory, especially of recent occurrences; and a lack of power of attention or concentration which prevents intelligent conversation.

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[1] Brissaud. Des Paralysies Toxiques, p. 31, Paris, 1886.

[2] Middleton Goldsmith Lectures, 1887. Medical News, March, 1887.

[3] Arch. f. Psych., 1892, Bd. xxi., 669.

[4] Rev. de Méd., May, 1897.

The patients are usually greatly depressed and alarmed at their condition, and require hourly reassurance of the probability of recovery, forgetting what has been told them almost as soon as it has been uttered. The depression may lead to crying for hours at a time, but the self-reproaches common in true melancholia are never heard in this state. The indifference to bodily wants may be so great as to lead to uncleanliness, and since paralysis of the sphincter is the rare exception, incontinence, if it occurs, is to be ascribed to the mental state.

It is useless to attempt to get any reliable history of their illness from these patients. Their statements are unintelligible or unreliable. They will relate occurrences as having happened recently, with much elaboration of detail, when as a fact the story is entirely a product of their imagination. Thus one patient of my own who had been confined to bed for many days, told me one afternoon that she had been out to see an eminent gynecologist during the morning; had gone to his office and waited for him several hours; had seen other patients there, and finally had been told by the doctor's brother that he would not return in time to see her, so she had come home again. And this was all related in apparent good faith, so that I have no doubt that she believed that what she said had occurred. Several patients have declared that they had been out to walk or to drive, when they had been unable to leave the bed for some weeks. One patient told me a different history of her case every day for a week, and it was only from her friends that the correct account was obtained. With the possibility of such delusions in view, it is evident that the statements of these patients cannot be accepted regarding anything, especially as to their own history.

The course of alcoholic neuritis is quite uniform. After a sudden onset the symptoms advance rapidly to a high degree, which is reached in a week or two from the beginning of the paralysis or ataxia. Then they may increase further, and cause death by respiratory paralysis. Usually they remain stationary for a time, which may be several months in severe types, and then gradually subside, the entire duration being from four months to two years. Individual muscles regain their power, tone, firmness, and electrical reaction slowly, and during recovery the tingling and numbness in the hands and feet may be severe. In a few cases the muscles become contractured, and permanent deformities develop, only to be overcome by long-continued massage or by operative measures. These deformities cause great difficulty in regaining the power of walking. The feet cannot be placed flat on the ground and the knees are partly flexed, so that it is often months before the patients can stand, and even when by the aid of high heels on the shoes, supporting braces to the ankles, and crutches, the upright position can be attained, it is with difficulty that the balance can be preserved. When the fact is considered that those who recover rapidly rarely fail to resort again at once to the use of stimulants, and thus expose themselves to the danger of a relapse, the ultimate fate of the chronic cases is hardly more serious than that of those who get well quickly.

The **treatment** of alcoholic neuritis differs in no respect from that already described on page 112.

The following cases illustrate the various forms of the disease:

A young man who had drank whiskey eight or ten times a day for some months without ever being drunk, began to feel a numbness in the legs and then in the hands, which was followed in three weeks by some undue fatigue on walking; occasional sharp pains in the legs, and a week later by weakness in the extensors of the feet and unsteadiness of gait. He went to the Hot Springs of Virginia, was treated for rheumatism, as he suffered while there from pains in the legs, but was finally told after two weeks that he had locomotor ataxia and was advised to go home. When I saw him on his return his subjective numbness was attended by a slight anæsthesia of the fingers and toes. His calves and forearms were very tender to pressure and the extensors were decidedly weak, so that he could not raise his toes from the floor when standing, but he had no drop-foot or drop-wrist. He was quite ataxic in his gait, swayed with eyes closed, but had no ataxia of the hands. His knee-jerks were absent, but he had no disturbance of his bladder, and his pupils reacted to light. His pulse was rapid and feeble, and he was subject to faint feelings and occasional sudden fears. He was put to bed, deprived of alcoholic drinks, fed frequently and well, and given two warm baths daily and general massage. Cod-liver oil and glycero-phosphate of soda, 15 grains, three times a day, were given daily, with strychnine, $\frac{1}{60}$, three times a day, four days in each week. Within four weeks his symptoms had improved so much that he was allowed to get up. At the end of nine weeks his paralysis had entirely disappeared and he no longer had pains or anæsthesia or any ataxia; and fourteen weeks from the date of his return from Virginia he was perfectly well, except for a very slight numbness in the sole of one foot. The knee-jerks returned in the tenth week. The entire duration of his illness was nineteen weeks. He has had no recurrence.

A man, aged thirty-six years, a hard drinker, after an attack of gastritis became paralyzed suddenly in both legs. A few days after this both hands became useless, so that he was completely helpless. On admission to the hospital, seven weeks after the onset, he complained of pain in all four extremities, and of tenderness in the muscles and joints when these were handled. The upper extremities were almost totally paralyzed below the elbows, the only motion possible being a slight flexion of the fingers. There was drop-wrist on both sides. The lower extremities were totally paralyzed below the knee, and the feet hung down motionless. The muscles of the thighs were also affected, for although he could pull his legs up in bed, it was with great difficulty that they could be straightened out. All the paralyzed muscles were atrophied, and showed a reaction of degeneration. Tactile sense was considerably impaired in areas corresponding to the region covered by long stockings and gloves, but the senses of pain and temperature and muscular sense seemed to be natural. The knee-jerks were lost. When an attempt was made to have him stand, the feet were pushed out and the knees doubled under him at once. Fibrillary twitchings were very noticeable in the atrophied muscles, and a marked tremor of the tongue was seen. The facial and ocular muscles were not affected, and the special senses were normal. There was no incontinence of urine, and no symptoms of thoracic or visceral disease. His mind was much impaired. He talked in a rambling manner, laughed much, and could not fix his attention, and his memory was so poor that no reliance could be put in his statements. Under treatment by complete cutting off of all stimulants and by iodide of potash he began at once to improve. In two months from the time of admission he was able to walk, and two months later he was discharged perfectly well. In this case the duration was six months.

A young woman, whose father died of alcoholism, had drank champagne and brandy very freely for two years during a period of mental distress. She was then (August) suddenly seized with paralysis attended by severe pain in arms and legs. She was at the time in Europe but was brought home, being carried to and from the steamer. When I saw her in November she had a typical alcoholic neuritis, with great tenderness along the nerves in the calves and on the front of the thighs, and drop-feet; cold, clammy hands covered with scales and pigmented, and anæsthesia in the glove and stocking areas. She was suffering intensely from such pains and hyperæsthesia in her arms and legs that she could not bear the weight of the bedclothes. She lay in bed with knees flexed and feet extended; the knee-jerks were absent. Her pulse was feeble and rapid and she was in a state of great mental distress at her condition. She required anodynes to relieve the pain. Under food, cessation of alcohol, which was extremely difficult to secure, as it was the only thing which relieved her pains, and general tonics, especially strychnine, she improved rapidly. In February she was able to walk with the help of high heels and corset shoes, and in April all her symptoms had disappeared excepting numb sensations and an occasional pain in her feet. Her knee-jerks had returned. She soon resumed drinking in spite of warning, and six months after her recovery she was again paralyzed; this time the hands as well as the feet being helpless, and drop-wrist developing at the same time as drop-foot. I saw her in December in the second month of this attack. She had a pulse of 150, irregular and intermittent, and was cyanotic. Her suffering from pain was extreme, and contractures existed in all the extremities. Glossy skin was present on legs and arms, and the extremities were hot and perspiring. Her mind was much affected. She had hallucinations of touch and sight, affirmed that her hands and feet were filled with needles, which she spent hours in drawing out of the skin, filling an empty box which she showed me as full of them; declared that scissors and knives were still buried in her flesh and cutting her. She had no memory of the events of the past two months. This condition continued for three weeks, her life being despaired of on account of her weak heart. During this time she told me daily some new story of having been to balls and receptions, and to drive in the park. Then (January) her circulation improved, her pains became less severe, she became less restless and was able to sleep, and her mind gradually became clearer. At the end of April she could use her hands a little and had lost the glossy skin and her mind was clear. But it was not until October that she was able to walk, and then she had to wear braces. In January she had recovered.

MULTIPLE NEURITIS DUE TO POISONING BY CARBON-MONOXIDE OR ILLUMINATING GAS.

A certain number of persons are brought into the hospitals every year suffering from acute poisoning by illuminating gas. Either accidentally or with suicidal intention they have turned on the gas in their sleeping rooms, and have been found after several hours in a state of coma. If they survive this condition of acute poisoning they sometimes develop symptoms of multiple neuritis within a week. These symptoms consist of tingling and numbness of the extremities, more acutely felt in the distal parts and attended by slight anæsthesia, also a condition of weakness in the muscles which does not go on to a total paralysis but makes them feeble. There are rarely any atrophies or changes in the electrical contractility in the affected muscles, and the sensory symptoms are, as a rule, much more marked than the motor symptoms. For many weeks, or even months, these patients suffer from paræsthesiæ which are excessively disagreeable. In a few cases I have seen a slight ataxia of movement, both in walking and in the use of the hands. The symptoms gradually subside under general tonic treatment, and the patient finally recovers.

The following case is of interest, as it appears to be a case of multiple neuritis following poisoning by natural gas:

The patient was a healthy man of good habits who had been very much exposed to the fumes of natural gas in his home in Peru, Ind. He gradually developed symptoms of tingling and numbness in the legs, which soon after extended to the hands and increased in extent until the legs and thighs and the hands and forearms were the constant seat of disagreeable numbness. After suffering in this manner for three months he began to notice an uncertainty of gait, which soon became a marked ataxia. There were at no time any pains, paralysis, atrophy, fibrillary twitchings, or disturbances of the bladder and rectum. His knee-jerks were normal and his pupils reacted perfectly to light, but his gait was distinctly ataxic; he swayed with his eyes closed and had a marked condition of anæsthesia to touch, temperature, and pain as high as the middle of the thighs and elbows, this anæsthesia being more intense in the hands and feet than in the upper parts. He had had during the entire illness some gastro-intestinal catarrh with attacks of diarrhœa, but careful investigation failed to reveal any possible source of poisoning by lead, arsenic, mercury, or drugs. It was discovered, however, that in both his house and office natural gas was burned for heating and lighting purposes, and that the fixtures were imperfect, resulting in incomplete combustion and leakage so that it was necessary to make special provision for ventilation, and both house and office were often filled with foul and nauseating gases when the draughts through the ventilators were imperfect. On removal from his home to the Clifton Springs Sanitarium, he rapidly improved under general treatment and was able to return home after three months quite recovered. A year later, however, also in the spring, after the confinement of the winter, the symptoms recurred with equal intensity and remained for eight weeks until he again left home, when, after four weeks, they subsided. In the absence of any other cause (and great care was taken to ascertain some cause for the condition) the neuritis was ascribed to the inhalation of natural gas.

A number of cases of neuritis in single nerves have been reported by foreign observers occurring after poisoning by illuminating gas. It is to be remembered that in a state of long-continued coma, pressure upon nerve trunks may occur of sufficient duration to cause traumatic neuritis which has not, under such circumstances, any relation to the cause of the coma.

MULTIPLE NEURITIS DUE TO POISONING BY SULPHIDE OF CARBON.

Multiple neuritis may develop in workmen who are exposed to poisoning from sulphide of carbon. This substance is present in rubber and the disease has been observed in those who work in rubber factories where ventilation is neglected. The fumes or the powdered dust containing sulphide of carbon are inhaled or get into the mouth and are taken into the stomach. The effect of the poison is slowly developed and seems to be widespread. In addition to a state of general anæmia, with disturbances of digestion, the patients suffer from headache, inability to fix the attention and to remember, and from many symptoms of functional nervous disorder, which may awaken a suspicion of hysteria; but as time goes on the symptoms of multiple neuritis appear, and these may be of the paralytic or of the ataxic type, and pursue a course not unlike that observed in alcoholic or arsenical cases. The development of cerebral and of spinal symptoms in some of the recorded cases makes it probable that the poison has an effect upon the central nervous system, and that the lesion is not in all cases limited to the peripheral nerves. Autopsies are wanting to establish an exact pathology.

The disease is exceedingly rare and has been described as a curiosity by various foreign observers.[1] I can find no cases published in this country, and this is very surprising, as the development of our rubber industries has been extensive in recent years.

MULTIPLE NEURITIS DUE TO POISONING BY THE COAL-TAR PRODUCTS, ESPECIALLY SULPHONAL AND TRIONAL.

Since the general use of the coal-tar products in the treatment of disease, especially of painful affections, and the free use of sulphonal and trional in the treatment of insomnia, a few cases of multiple neuritis have been reported, occurring in patients who had abused these drugs. And their chemical resemblance to alcohol lends a certain amount of support to the hypothesis that they are capable of producing multiple neuritis. Remak has reported a case as having occurred subsequent to the use of antipyrine, and Stewart Hart[2] has recorded a case, which I saw with him, probably produced by the constant use of trional. A patient of my own, who took 120 grains of sulphonal in four hours by mistake, slept two days, was markedly cyanotic, with pulse of 120 and shallow respiration, and, for three weeks afterward, suffered from paræsthesiæ, weakness, and ataxia in the arms below the elbows and in the legs below the knees. The symptoms gradually subsided. They resembled so closely those which I had seen in hospital cases of illuminating-gas poisoning, that it seemed justifiable to ascribe them to a mild condition of neuritis.

Multiple neuritis has been said by Ross to have been caused by poisoning with roburite (dinitrobenzol) used by miners, and by poisoning with anilin oil, used by dye manufacturers. In the cases recorded the symptoms in both forms of poisoning were those of multiple peripheral neuritis, together with general constitutional effects—anæmia, cyanosis, and a blue discoloration of the mucous membranes, due to the action of the poison on the hæmoglobin.

ARSENICAL NEURITIS.

Historical. Up to the year 1883 the various nervous affections produced by acute or chronic poisoning by arsenic had been ascribed to spinal-cord lesions. Vulpian,[3] Virchow and Scolozouboff[4] insisted upon the central origin of arsenical paralysis; the latter, with Popow,[1] basing his assertions upon physiological experiments. Dejerine,[2] however, in 1883, after his studies of alcoholic paralysis, already cited, concluded from the close resemblance between alcoholic and arsenical paralysis that the lesion was a multiple neuritis. And subsequent observations by Philadelphia[3] and Boston physicians, by Jaesche,[4] Naunyn,[5] Dana,[6] Goldflam,[7] Jolly,[8] and Henschen,[9] have established beyond doubt that the lesion in arsenical poisoning lies exclusively in the nerves. This fact has received complete confirmation during the prevalence of an epidemic of arsenical poisoning from beer which occurred in England in 1899.[10]

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[1] R. Landenheimer. Die Schwefelkohlenstoff Vergiftungen der Gummiarbeiter. Leipzig, 1899, where full references to all published cases, about forty in number, can be found.

[2] American Journal of the Medical Sciences, 1901.

[3] Leçons sur les Mal. du Syst. Nerv., 1879, p. 157.

[4] Arch. de Phys. Norm. et Path., 1884, p. 325.

Etiology. Arsenical poisoning may occur from the accidental or suicidal ingestion of any one of the arsenical salts—Paris green being the one usually selected by those attempting suicide. The most common cause, however, is the accidental chronic poisoning which occurs in certain manufactures, notably in tin mines, and in tin works; in dyeing establishments and in sulphuric acid factories, and in those industries in which sulphuric acid is largely used. This acid is now made chiefly from arsenical pyrites and thus contains considerable quantities of arsenic.

Chronic arsenical poisoning may occur from drinking beer in the brewing of which glucose, or "invert sugar," prepared by the aid of sulphuric acid, enters. Thus in the English epidemic it was ascertained that many beers brewed in Salford and Manchester contained from 0.14 to 0.28 grain of arsenious acid to the gallon, and this was shown conclusively to be present only in the glucose or invert sugar used in the brewing, all other constituents of the beer being free from arsenic. The glucose was found to contain arsenic in the proportion of 4 parts to 10,000.

Chronic arsenical poisoning may also arise from the inhalation of particles of arsenic in the air, which are given off from wall-papers and from carpets, hangings and furniture coverings, and from artificial flowers containing arsenical dyestuffs.[11] This was the cause in a case of my own. The patient was a woman, who, to avoid miscarriage, was kept for four months in a room which had been newly papered and furnished with green cretonne subsequently found to contain large quantities of arsenic. The frequency of such poisoning has led to the passage of laws in the State of Massachusetts restricting the use of dyes containing arsenic. Many toilet powders contain arsenic, and the inhalation of particles given off from these, or the application of arsenic in them to the skin may produce poisoning. The medicinal use of arsenic in large doses or in small doses long continued may give rise to arsenical poisoning. This is particularly liable to occur in the treatment of chorea, of pernicious anæmia, and of carcinoma, in all of which diseases the drug is employed for months at a time. I have seen two cases of arsenical paralysis among 1400 children treated for chorea in my clinic. The more the danger of chronic arsenical poisoning is appreciated and the greater the use of articles into the manufacture of which it enters, the more frequent are the reports of cases arising in an unexpected manner. It is known that in Steiermark, in Austria, large numbers of the peasants eat arsenic. F. Müller[1] has found arsenical paralysis very common among this class, in fact, even more common than diphtheritic paralysis.

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[1] Virchow's Arch., 1883, Bd. 93, p. 351.

[2] Comptes-rendus, Tome xcviii., Nr. 17, Oct. 1883.

[3] Proceedings of the College of Physicians of Philadelphia, 1883.

[4] Inaug. Dis., Breslau, 1883.

[5] Berliner klin. Woch., 1886, p. 555.

[6] Brain, 1887, vol. ix. p. 456.

[7] Zeitsch. f. klin. Med., 1888, p. 399.

[8] Deutsche med. Wochen., 1893, Nr. 5, and Charité Annalen, 1893.

[9] On Arsenical Paralysis, Transactions of the Royal Society of Sciences in Upsala, 1893.

[10] Kelynack and Kirkby, Arsenical Poisoning in Beer Drinkers, Balliere, Tindall and Cox, London, 1901. Also Lancet, 1900, vol. i. p. 1610.

[11] J. J. Putnam. Boston Medical and Surgical Journal, 1889, p. 235.

Pathology. A simple parenchymatous neuritis, without any interstitial inflammation, has been found in many cases of arsenical multiple neuritis. In these cases the chief change is limited to a destruction and degeneration of the myelin sheath, the axis cylinder remaining intact. The myelin is broken into fragments or little balls, which are found in all stages of fatty and granular degeneration within the sheath of Schwann. The nuclei within the sheath are increased in number. This lesion has been termed segmental periaxillary neuritis, as the process may be scattered along the nerve and may not be continuous from one segment to the next. In severer cases the axis cylinder is destroyed, being found in all stages of degeneration. In these cases many empty sheaths of Schwann are seen.

Changes have also been found in arsenical paralysis in the spinal cord. The same chromatolysis in the cells of the anterior horns, which has been described in alcoholic paralysis, has been observed, but this, as already stated, is of little importance. Henschen has found a true degeneration in these cells, and has also noticed an ascending degeneration in the columns of Goll, which may be due to a primary degeneration in the cells of the posterior spinal ganglia.

Symptoms. The symptoms of arsenical neuritis do not differ very markedly from those of alcoholic neuritis. In both there are two types of case—one characterized by paralysis, and the other by ataxia. There is little or no difference in the mode of onset of the symptoms between cases of acute and chronic poisoning.

There are certain general constitutional symptoms which first appear in arsenical poisoning and attract attention. If the poisoning is acute, violent vomiting and diarrhœa occur at once, and sometimes save the life of the patient, the poison being rejected and not absorbed. If the poisoning is chronic, a puffiness of the eyelids appears, at first most noticeable in the morning; a tearful appearance of the face, and some catarrh of the nose is observed, and not infrequently gastric and enteric symptoms develop. There may be pain in the stomach, nausea and inability to retain food; and fluid, green or slimy movements with some tenesmus. In some cases a mild fever attends the attack, a rise of 1° or 2° F. occurring and persisting for some weeks.

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[1] Wiener med. Presse, 1894, Nr. 15.

In arsenical neuritis the first symptoms complained of are sensory in type. Tingling and numbness in the feet and hands, burning of the surface, tearing and shooting pains, and great soreness in the skin and in the calves of the legs and feet, and soon a feeling of weakness in the legs, great fatigue on walking, difficulty in rising from a seat or in going up stairs, and a staggering gait are noticed. As the general feebleness increases, paralysis develops rapidly in some cases, in others ataxia is more marked. The paralysis appears in the feet and hands, the lower extremities being first affected. There is the same limitation of this paralysis to the small muscles and to the extensor groups of muscles of the extremities that is seen in alcoholic cases. The paralysis is flaccid in type, is soon followed by atrophy, which is first noticed in the interossei of the hands, and then appears in the peronei of the legs and in the extensors of the wrists, and is attended by loss of muscular tone and mechanical excitability. The reaction of degeneration is usually present. The tendon reflexes are diminished or lost. The superficial reflexes are normal or are increased. Drop-wrist and drop-foot develop early and may become extreme. The distribution of the paralysis is always bilateral and symmetrical. In severe cases the patients are confined to bed, and contractures in a flexed position gradually develop and may become permanent. Thus in a case of mine which followed acute poisoning, the hands were fixed in a claw position for many months after all other symptoms had passed away.

When the ataxia precedes paralysis the gait becomes unsteady and irregular, the patient sways in standing with eyes closed, he drags his feet and shuffles along and finally shows the "stepping gait" described in alcoholic cases. The dropping of the feet serves to distinguish this gait from that of tabes, though French writers have described these cases as *pseudo-tabes arsenicale*. Such a gait was well marked in both patients with chorea, who developed arsenical paralysis under my observation. The ataxia is also present in the hands and is accompanied by an impairment or loss of muscular sense. It is always attended by a considerable degree of weakness, and this commonly goes on to paralysis and the patients have to go to bed.

In both types of the disease, when the symptoms are fully developed, the pain and tenderness are extreme. Many patients cannot endure the slightest touch, even the bedclothes causing agony; and the extreme tenderness in the muscles below the knees and elbows prevents any manipulation of the extremities. The hyperæsthesia is felt more acutely in the distal parts of the extremities and rarely above the elbows and knees, but when the ataxia and paralysis are fully developed it renders the patient particularly helpless. A marked tremor in the hands is usually an early symptom in both types of case. The ocular muscles and the facial muscles are not affected and the trunk muscles, the intercostals, and the diaphragm also escape. Occasionally erythema, redness of the skin, œdema of the extremities, cyanosis and unusual sweating, indicate an implication of vasomotor and trophic nerve fibres. Some observers have reported a condition resembling erythromelalgia.

The diagnostic symptom of greatest importance in arsenical neuritis is the appearance presented by the skin. This becomes gradually darker from the deposit of pigment, so that the complexion resembles that of a gypsy, and if extreme the skin may finally turn almost black. The pigment is first deposited about the normally pigmented regions of the body, in the axillæ and groins and around the nipples. It soon appears, however, on the eyelids, on the abdomen and thorax, and finally on the surfaces of the arms and legs, the flexor surfaces being last affected. The pigmentation is diffuse, but here and there little islets of normal skin give a mottled appearance to the surface. The color is rather darker than that seen in Addison's disease, and the mucous membranes are never discolored as in that affection. The pigmentation may be preceded by erythema and may be followed by various eruptions, papillary, eczematous, or herpetic. Herpes has been observed in many cases, usually on the limbs, sometimes on the body. After a time the skin desquamates in small flakes, but several desquamations may be necessary before a normal color is regained. Sometimes there is a marked thickening of the skin, especially over the knuckles, on the palms and soles. Changes in the nails are very common, a thickening and brittle state being evident and rough ridges appearing transversely. The appearance of normal nails growing out above the ridges indicates beginning recovery. In the Manchester epidemic there were skin lesions in 97 per cent. of the cases.[1]

In one case of acute poisoning by Paris green, a reddish eruption appeared on the tenth day upon the hands, wrists, and forearms, and upon the ankles and feet, and in twenty-four hours had spread over the entire body. Four days later this had gradually disappeared, and then numbness was first felt in the hands and feet. This extended rapidly, and in three days had reached elbows and knees. Paralysis began at the same time as the numbness, and in a week all power of movement below the knees and elbows was lost. This remained for two weeks, and then slowly passed off, recovery not being complete for six months.

The sphincters are never involved in arsenical paralysis.

Mental symptoms have been described by a few observers, consisting of hallucinations, mild delusions, stupor, and loss of memory. In the English epidemic these were observed only in individuals who showed other signs of chronic alcoholism, but Jolly has seen them in a non-alcoholic case. And the fact that arsenic has a stimulating action upon the brain is said to be one reason for its use as a food by the Steiermark peasantry.

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[1] British Medical Journal, 1900, vol. ii. p. 1725.

The symptoms all subside slowly and progressively, and recovery of power and of co-ordination is accompanied by a relief from the hyperæsthesia and the pain.

The duration of arsenical paralysis is somewhat greater than that of arsenical ataxia. Either condition may last several months, but occasionally the recovery is complete in a few weeks. Frequently, after the power has returned, the patient suffers for months from numbness and tingling in the extremities, which are sufficient, as in a case under my own observation, to interfere with the finer motions, to disturb the sleep, and to cause constant discomfort. According to Gerhardt, 97 per cent. recover wholly. And in the English epidemic, although the number of cases was very large, several thousand having been observed in the hospitals and dispensaries, the number of deaths reported was comparatively small.

Treatment consists in elimination of the arsenic by the free use of water and in restoring the general health of the patient. It does not differ in any way from the line of treatment for multiple neuritis, already described in detail on page 112.

The following case is interesting on account of its causation, and offers a typical history of the disease:

Female, aged thirty years, previously in good health, became pregnant in November, 1894, and until March 10, 1895, suffered exceedingly from the vomiting of pregnancy, and became rapidly emaciated from inability to retain food, so that she lost ninety pounds in weight. During this time she was confined to one room, the furniture and wall-paper of which on subsequent examination were found to contain about two grains of arsenic to the square yard. On March 10th she began to suffer from tingling and numbness in the legs and noticed some difficulty in stretching out her legs, and this paralysis increased rapidly, so that by the 15th of March there was almost total paralysis of both legs and very great pain and tenderness in both legs, together with a sense of numbness and tingling in the feet and legs as high as the knees. There was great sensitiveness of the legs to cold and heat, and any movement of the legs was extremely painful, the pain being felt in the muscles. The knees were preferably kept in a position of flexion as extension was too painful to be endured. On the 20th of March an extensive eczema appeared all over the body, but more particularly in the legs and arms, and was attended by a deep brown pigmentation of the skin and by some desquamation. On April 1st, a total paralysis had developed in the feet and ankles and drop-foot was present on both sides. The muscles below the knees were very weak, but the thighs could be moved normally. The knee-jerks were lost. Any attempt to stand was impossible on account of the paralysis of the feet and ankles, and the extreme pain that any pressure upon the soles of the feet produced. The muscles were flabby and the muscular tone and mechanical excitability were abolished, and faradic contractility was lost in the muscles below the knees. There was no affection of the bladder or rectum and no tendency to bed-sores, but the nails of the toes were discolored and ridged, and the nails ceased to grow for three months. There was a constant sensation of tingling and numbness in the fingers, but no evidence of paralysis or anæsthesia. Her room was changed, thus removing her from the source of the poison. The gastro-intestinal catarrh, which was doubtless due to the arsenic, soon subsided under treatment and she began to retain her food. In August she was able to walk with a little aid. In December she still required some help in walking, as the foot-drop persisted, and braces had to be used for the ankles, with high-heeled shoes, and at this time her legs were still stiff and numb. There were no knee-jerks, and sensations of pain, heat, and cold were much less quickly and keenly perceived below the knees than above, or upon the hands. In April, 1896, she had recovered entirely.

MULTIPLE NEURITIS DUE TO POISONING BY LEAD.

Etiology. Poisoning by lead is always a chronic poisoning and usually occurs in painters, in plumbers, in typesetters, or in workers in lead objects such as toys, who come in contact with lead, either in the form of metallic lead or of some of the salts of lead which are used in mixed paints. Painters who work in the open air are less liable to lead poisoning than those who labor in rooms; and those are most liable who, like the varnishers in carriage factories and furniture establishments, scrape off old paint preparatory to putting on a new coat, Any work which results in the production of dust which may be mixed with lead, or its salts, may be productive of lead poisoning. It is evident that the inhalation of the dust is one method of the entrance of the poison into the body. Laborers who work in factories where china earthenware is produced and where glazes and fretted glass are manufactured are very liable to lead poisoning. In the midland counties of England, where such industries are common, in Limoges, France; in Dresden, Germany; and in Trenton, New Jersey, many cases are seen annually.

The usual method of poisoning, however, is by swallowing the lead, and, as many painters do not properly clean the hands before taking their noonday meal, the poisoning is often acquired in this way. Some individuals, however, appear to be extremely susceptible to lead poisoning. Thus, I have known most serious cases in children of painters who could only have obtained the poison by inhalation of the fumes of paints that were kept in the room. I have known small amounts of lead to produce severe poisoning when taken into the stomach. Beer bottles are frequently cleaned by being shaken with lead shot within them, and I have known lead poisoning to occur from drinking the beer from such bottles. I have known lead poisoning to occur in servants who drank the water first drawn from the spigot early in the morning, water which had stood in the lead pipes all night and had thus become impregnated with some of the soluble salts of lead. I have known severe cases in typesetters whose fingers were stained by the constant handling of metallic lead. I have also known it in plumbers after long labor in laying lead pipes. Horses are subject to lead palsy. Lead is not frequently used as a medicine, and I have never known a case of poisoning from this source. Alcoholic subjects are more liable to develop lead palsy than others.

Pathology. The pathology of lead palsy has been a matter of dispute among authorities for many years. Many writers have found changes in the cells of the anterior horns of the spinal cord, while others, more recently, have maintained that the changes are limited to degeneration of the peripheral nerve trunks. The establishment of the neurone theory has thrown light upon these differences of opinion, and the discovery of more delicate methods of staining by Nissl and Marchi have demonstrated that the entire motor neurone may be affected in this disease. The most evident changes are a degenerative neuritis with atrophy in the peripheral nerves, chiefly in the radial nerve, but not infrequently in many of the peripheral branches of the brachial plexus. The degree of this degeneration and its extent depends upon the severity of the poisoning. There are few, if any, changes in the neurilemma or connective tissue sheath, hence this form of neuritis is purely a parenchymatous neuritis. While the majority of these changes are limited to the terminal filaments of the nerves, there may be slight changes detected in the trunk of the nerves and in the anterior nerve roots. The method of Nissl demonstrates chromatolysis in the cells of the spinal cord and in those of the spinal ganglia, but these are not usually of a sufficient degree to produce degeneration or complete atrophy that is irreparable. In the severer cases, however, vacuolization of the cell and final degenerative changes may take place. Changes of a similar kind have also been found in the large cells of the brain cortex. The serious lesions are more commonly found in the peripheral nerves than in the spinal cord.

Symptoms. In 93 per cent. of cases the onset of the paralysis is preceded by a severe attack of lead colic. The patient suffers from intense pain in the region of the umbilicus, attended by obstinate constipation, very often by nausea and vomiting. The pain is supposed to be due to an irritation of the visceral nerves by the lead. It may also be due to distention of the intestine from paralysis of these nerves and cessation of peristaltic action. There is a characteristic blue line seen along the gums, not to be mistaken for staining of the teeth because of its location and clear blue color. Anæmia is almost always present in patients who have been poisoned by lead. Another common affection in persons exposed to lead poisoning is chronic articular rheumatism. Thus, Tanqueral found that in 1217 patients who suffered from lead colic, 755 developed painful joints, 107 developed paralysis, and 72 suffered from headache.

A few days after the onset of an attack of colic, or sometimes not until two or three weeks after the attack has subsided, the patient is suddenly seized with paralysis of the extensor muscles of the fingers, thumb, and wrist. It is a rule for one hand to be affected several days before the other, and occasionally both hands are unequally affected throughout the disease. It is quite commonly found that the paralysis of the extensors of the index finger or of the thumb precedes the paralysis of the other muscles. As a consequence of this paralysis the wrists drop and cannot be raised, and from lack of fulcrum power the flexors are apparently weakened. If, however, the wrists be extended by the examiner and held, it will be found that the flexor power in the hand and fingers is not impaired. The supinator longus muscle is rarely, if ever, affected, and hence it is apparent that the lesion is limited to the distribution of the radial branch of the musculospiral nerve below the point where the nerve to this muscle is given off. There is very often a tremor in the hands and fingers, which comes on early and remains until the paralysis subsides. There is no affection of sensation as a rule, though sometimes tingling and numbness is felt in the back of the hand or in the fingers. The muscles that are affected rapidly undergo atrophy of an extreme type, so that within a couple of weeks of the onset the back of the forearm presents a very atrophic appearance and the muscles show the reaction of degeneration.

As a result of the drop-wrist a deformity of the back of the hand soon appears, due to a displacement backward of the internal bones of the wrist, forming a hard protuberance upon the back of the hand near the base of the metacarpal bones; but firm contractures or permanent shortening in the paralyzed muscles, such as occurs in alcoholic or arsenical cases, does not develop in lead palsy. Not infrequently, if the disease is a severe one, the intrinsic muscles of the hands also suffer early and the hands become quite useless, apposition of the thumb to the fingers and any abduction or extension of the thumb and fingers being impossible. Then it is evident that branches of the ulnar and median nerves are also involved.

The reaction of degeneration appears early in the muscles, and the faradic reaction does not return until the patient has been well for some months.

In the majority of cases the paralysis does not go beyond the muscles of the forearms and hands, and after a period of five to six months there is a gradual return of power and complete recovery. The patients often complain of severe cramps in the muscles—a form of pain which may even prevent sleep—but pain is not a common symptom in lead palsy.

The disease is a very slow one and the patients are rarely able to return to work within six or seven months of the onset. As their livelihood depends upon their labor, it is well that this should be understood from the outset, so that during the period of incapacity they may seek some other occupation which will enable them to support life. Relapses are common when patients return to their work.

In more serious cases the paralysis may appear in the muscles of the shoulder, the deltoid being first affected, and then the biceps, brachialis anticus and supinator longus being involved. Occasionally the muscles of the legs below the knees become paralyzed, the peroneal group being more commonly invaded, though in severe cases the extensors of the toes and even the anterior tibial group may be equally paralyzed. This only occurs in 4 per cent. of the cases and has been observed chiefly in children. It causes drop-feet and a stepping gait. In the most severe type the patient is almost totally paralyzed. Fibrillary twitchings are occasionally seen in the muscles but they are not the rule. A general sensory disturbance is common, the patients suffering from intense pain in the arms and legs, from anæsthesia of the hands and feet, and from very disagreeable sensations of pricking and tingling. The sphincters are never involved. Vasomotor and trophic symptoms are usually absent, though the hands and arms may be cold, cyanotic, and may perspire too freely.

In the severe cases it is possible to demonstrate a condition of tenderness along the affected nerves, but in slighter cases no tenderness is felt. A type of lead palsy has been described in which the symptoms appear first or exclusively in the cranial nerves; the laryngeal, the pharyngeal, the lingual, the ocular, the facial, and the optic nerves having been affected. In these cases headache, vertigo, and mental apathy indicate that the brain as well as the nerves is invaded by the poison. This type is very rare. It is termed lead encephalopathy by the German writers.

Prognosis is good as to recovery, but the duration of the disease, as already stated, even in the lighter type, is six months, and in the more severe type very often a year. The following case illustrates a severe condition of chronic poisoning:

Male, aged thirty-eight years, after suffering from an attack of lead colic, began to feel numbness and pain in his legs and feet, which soon extended to his hands and forearms. This steadily increased for two weeks, and to it was added paralysis of the extensors of both hands and both feet, so that at the end of that time he was unable to use his hands or to stand. With the paralysis there was rapid atrophy of the muscles, and a decline in the faradic excitability. The muscles became more and more tender, and the spontaneous pains gradually increased until it was necessary to use opium freely to quiet him. By the end of a month a well-marked anæsthesia had developed in the hands and below the knees, and it was noticed that the atrophied muscles were in a constant tremor. There were wrist-drop and foot-drop on both sides, and all reaction to faradism ceased. It required a very strong galvanic current to produce contraction and ACC was greater than KCC. The knee-jerk was preserved.

The muscular sense was impaired. For five months his condition remained stationary, in spite of treatment by strychnine, iodide of potash, massage, and electricity. During a month of his illness he was delirious at night, was excited in the daytime, and subject to delusions and had little memory or mental capacity; but these symptoms gradually subsided. Then a gradual improvement set in, the pains became less severe, sensation returned, the muscles regained their contour and strength, and, finally, the electric contractility returned to the normal standard. About a year from the onset of the symptoms the recovery was complete.

Treatment. Treatment of the lead colic is by means of full doses of opium to quiet the pain, and by laxative saline salts, such as sulphate of magnesia, to relieve the constipation. Large draughts of water should be given constantly, in order to eliminate the poison, which passes off partly through the kidneys. During the stage of chronic poisoning the patient should be instructed to drink a great deal of water, and five grains of iodide of potash may be given three times a day to assist in the elimination of the lead. I have never been able to see that strychnine, which is usually recommended, has any effect upon the progress of regeneration, though general tonics —cod-liver oil, iron, and quinine—may well be employed, as these patients are uniformly anæmic. Treatment of the paralysis is by massage and electricity, galvanism being the only current which will produce any effect.

Multiple neuritis of the ataxic type, due to poisoning by phosphorus, has been recorded by Henschen in one case, and in seven other cases of acute poisoning he found mild nervous symptoms of paræsthesia and tenderness along the nerves. The condition must be a very rare one, as it has not been seen by others. Inquiry among employés in match factories failed to elicit any knowledge of a special disease peculiar to this occupation.

Mercurial poisoning has been said to cause multiple neuritis, but no case can be found which is not open to objection. The tremor which is produced by acute mercurial poisoning, and which is observed in workers in looking-glass factories, is accompanied by headache, vertigo, and occasionally by hemiplegia or monoplegia of cerebral type, and is not followed by multiple neuritis. Chronic mercurial poisoning after or during the treatment of syphilis also fails to cause neuritis.

Gowers[1] has described a case of poisoning by silver, in which paralytic symptoms developed in the arms with drop-wrists, resembling closely those due to lead poisoning. The characteristic blue coloring of the skin was present. The patient died of cancer and no autopsy was made. This is the only case thus far observed.

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[1] Diseases of the Nervous System, German edition, vol. iii. p. 335.

A few cases have been recorded[1] of multiple neuritis due to poisoning by copper occurring among brass workers. In these cases the symptoms resembled those of the ataxic type of neuritis seen in alcoholics. They are so rare as to require mention only.

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[1] Suckling. British Medical Journal, 1888, vol. ii. p. 1334. Walton and Carter, American Journal of the Medical Sciences, 1892, vol. ii. p. 61.

CHAPTER VII.

THE INFECTIOUS AND TOXÆMIC FORMS OF MULTIPLE NEURITIS.

Multiple Neuritis Subsequent to Diphtheria; to the Grippe; to Typhoid, Typhus, and Malarial Fever; to Scarlet Fever, Measles, Whooping-cough, and Smallpox; to Erysipelas and Septicæmia; to Gonorrhœa and Puerperal Fever. Leprous Neuritis.

MULTIPLE NEURITIS SUBSEQUENT TO DIPHTHERIA.

DIPHTHERITIC paralysis is one of the most common forms of multiple neuritis, but does not occur as frequently as a sequel in cases that are treated by antitoxin as in those which are allowed to run a normal course. The number of cases of diphtheritic paralysis appearing at my clinic in the past three years, since the use of antitoxin has been much less than in any previous period of three years. Rothe[1] found 64 cases of diphtheritic paralysis in 744 cases of diphtheria treated at the Charité Clinic in Berlin, which gives a frequency of 8.7 per cent. of the cases of diphtheria followed by paralysis. This observation is in accord with that of other observers.[2] Bernhardt called attention to the fact that in many cases of diphtheria there is a loss of the knee-jerk, a fact which seems to him to point to a probable susceptibility of the entire nervous system to the diphtheritic poison, but Rothe found a loss of the knee-jerk in but one-half of the cases which were paralyzed. It is certainly a fact that for many weeks after the disappearance of all symptoms of diphtheria, even in cases which show no paralysis, the knee-jerk may be absent.

Pathology. The pathology of diphtheritic paralysis has been a matter of much discussion. Some authors maintain that the lesion is a degenerative neuritis only, while others maintain that the lesions are central in the spinal cord and brain axis. A parenchymatous neuritis is the chief lesion, but occasionally a diffuse process with involvement of the interstitial tissues has been observed. In the finest nerves of the muscles and in the skin, the destructive process is more fully developed, as a rule, than in the trunks of the nerves or in the larger nerves. The neuritis is more pronounced in the cranial nerves in diphtheritic paralysis than in any other form of multiple neuritis. Lesions of the anterior and posterior nerve roots have been found by Dejerine, and many authors have described parenchymatous degeneration in the anterior horn cells and in the posterior spinal ganglia.

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[1] Inaug. Dissert, Berlin, 1899.

[2] Wollacott. Lancet, August 20, 1899.

Recent experimental investigations by Murawjeff[1] show that the toxin of diphtheria produces chromatolysis and degeneration in the cell bodies of both motor and sensory neurones. He also finds that it attacks the peripheral nerves as well; in some cases before, in others after it has attacked the neurone.

Symptoms. In the lighter forms of diphtheritic paralysis the soft palate is the only part that is affected. In 472 cases of diphtheritic paralysis observed by Wollacott the soft palate was affected in 413, the difficulty of swallowing, with regurgitation of fluids through the nose and a thickening and indistinctness of the voice being the only symptoms present. There is a loss of reflex in the throat in these cases and a lowered sensitiveness of the pharynx, so that irritation does not cause acts of swallowing. The limitation of the paralysis to the palate has been explained by supposing that the poison of the disease has a direct action upon the terminal filaments of the nerves which, in this position, are, as it were, dipped constantly in the poison. This theory is supported by a case in which paralysis of the abdominal muscles was associated with a diphtheritic inflammation of the navel in a newborn child. But the more serious cases prove that it is through the blood that the poison is carried to nerves far removed from the diphtheritic inflammation.

Next in frequency to paralysis of the palate occurs paralysis of some of the ocular muscles, producing internal squint and double vision. This paralysis may affect any one or many of the muscles moving the eyeball of one or both eyes, though it is less common for the levator palpebræ to be affected than any other muscle. The external recti are more often affected than the internal, and it is not common to find all the muscles supplied by the oculomotor nerve affected together. Among Wollacott's 472 cases, 104 had some ocular palsy. The paralysis of accommodation prevents reading. The paralysis may not extend to any other muscles than those already named, and in the lighter cases remains in them for a period of four to ten weeks and then gradually passes off.

Not infrequently, however, the disease extends much more widely and after a week or more of local palsy of the throat or eyes, or both, the individual develops within twenty-four hours a widespread paralysis of both arms and both legs. This is usually preceded or attended by incoordination of movement in all the finer adjusting actions of the hands and by ataxia in the act of walking. Associated with this ataxia and with the weakness of the muscles there are frequently drop-wrist and drop-foot, so that the patient exhibits the stepping gait already described as occurring in alcoholic neuritis.

There are also sensory symptoms consisting of numbness in the extremities, disturbance of sensation both of touch, temperature, and pain and of the muscular sense and delayed sensations of pain. Shooting pains are sometimes felt, but are not very prominent and there is rarely tenderness along the nerve trunks. If the paralysis is not so extreme as to suspend all movement, a tremor is not uncommonly observed in the motions of the fingers. The tendon reflexes are lost and mechanical excitability of the muscles disappears; a rapid atrophy with reaction of degeneration develops in the paralyzed muscles. Paralysis of both sides of the face may occur.

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[1] Neurolog. Centralbl., 1898, p. 475.

In a still more severe type of case, either soon after the onset or after the paralysis has been present for several weeks, bulbar symptoms may appear, with great difficulty in swallowing, atrophy, and weakness of the tongue, spasms of coughing (which may be dangerous), weakness of the face, and paralysis of the vocal cords. Such a complication is extremely grave, as the patient may die of respiratory paralysis, of suffocation, or of pneumonia due to the inhalation of food. This occurs in 10 per cent. of the cases. The onset of the paralysis may occur within a week of the infection or it may be delayed for several weeks. In Rothe's cases the paralysis developed in the majority in the second week, but in a few over forty days had elapsed between the initial symptoms and the development of paralysis.

Diphtheritic paralysis is not by any means confined to children, but occurs in adults and even in old people. Not infrequently it is impossible to ascertain a definite history of diphtheria, and a very light attack of diphtheritic sore throat may be followed by a severe attack of diphtheritic paralysis. The occurrence of paralysis of the throat and eye muscles and a marked ataxic condition of the limbs enable one to make a diagnosis, even in cases where no sore throat has occurred.

Prognosis. The prognosis in diphtheritic paralysis is good, provided the condition is limited to paralysis of the throat, or of the eyes, or to a general diphtheritic ataxia. If, however, a true paralysis of the muscles of the limbs ensues; if the respiratory muscles are affected, or if the pneumogastric nerve is invaded, causing frequency of the pulse, the prognosis is very serious.

Treatment. The treatment of diphtheritic paralysis is that of multiple neuritis. The danger of inhalation of food or of imperfect nutrition from difficulty of swallowing is not to be forgotten, and in the extreme cases it may be well to feed the patient with a stomach tube rather than to allow them to attempt to swallow, if this be productive of fits of choking and coughing. Inasmuch as all effort seems to increase the severity of the symptoms, it is well that these patients should be kept in bed as long as there is any difficulty of motion. Strychnine is of a great deal of service in the treatment of diphtheritic paralysis. It was for a time supposed to be a specific for the disease, but this was at a time when the lesion was thought to be central. It is not to be used in very large doses, $\frac{1}{100}$ of a grain every three hours being sufficient for an adult. It has not seemed in my experience to shorten the duration of the paralysis.

The following cases are good examples of the ordinary type of diphtheritic paralysis:

Male, two and a half years of age, suffered from measles during the month of January, 1894, and on the 1st of February developed symptoms of diphtheria. This ran its ordinary course, though it was necessary to perform intubation and the tube was retained about one week. He then gradually recovered and after a week appeared to regain his ordinary strength; his appetite improved, and he could stand and run about. On the 20th of March he was suddenly seized with paralysis, which within two days had become most extensive. He then had extreme strabismus due to a paralysis of both internal recti. There was no paralysis of the face, tongue, or palate, and he could talk well; but there was complete paralysis of the muscles moving the head and of both arms and both legs, and of the body. He was unable to sit up, to move his head, or to move any of the extremities. There was no knee-jerk. There was no disturbance of the bladder or rectum, there was no pain or tenderness; but respiration was difficult and his pulse was 140. On the 24th of March he died of respiratory paralysis.

A boy, aged thirteen years, after an attack of diphtheria developed paralysis of the soft palate. One week after the paralysis of the palate he noticed a disturbance of vision which proved on examination to be due to a total paralysis of all the muscles of the eyeballs. The pupil reflex to light was preserved; but there was no reaction in accommodation. This paralysis of the eyeballs came on within twenty-four hours, and at the same time he developed a very marked degree of ataxia. He swayed in standing with eyes closed, and his gait was exactly like that of a well-marked case of tabes. The motions of the hands were also disturbed, and all the tests demonstrated an extreme degree of incoordination. The knee-jerks were lost; the skin reflexes were present. In addition to the ataxia there was actual weakness of the muscles, which was more evident in the arms and hands than in the lower extremities. He had formication in the legs, but not in the hands, and there were no lightning pains. He had great difficulty in swallowing solids as well as liquids, which seemed to point to some involvement of the muscles of deglutition as well as of the soft palate. This condition remained stationary for about three weeks, and then all the symptoms began to pass off gradually. His recovery was not complete until six months after the onset.

A girl, aged thirteen years, after suffering for a few days from a slight sore throat, found herself unable to see clearly because of the development of a paresis of accommodation. There followed a slowly progressing ataxia of the upper, and later of the lower, limbs, with uncertain gait, disturbance of touch, pain, temperature, and muscular senses, with delayed sensation of pain and loss of tendon reflexes. At first she felt shooting pains in the limbs, but later these ceased. There was at no time tenderness of the nerves to pressure. After three months bulbar symptoms appeared, atrophy with paresis of the tongue, difficulty in swallowing, spasms of coughing, weakness of voice, and paralysis of the palate. The muscles of the hands began to atrophy about the time that the bulbar symptoms commenced and soon were useless. In the atrophic muscles the faradic reaction was absent, the galvanic much reduced and very slow. Nine months after the onset she died of pneumonia.

The autopsy showed the brain and spinal cord to be normal. In all the peripheral nerves of the extremities, as well as in the hypoglossal and recurrent laryngeal nerves, well-marked atrophic degeneration was found, with destruction of axis cylinders and medullary sheaths, and with thickening and increase of nuclei in the connective-tissue sheaths.[1]

MULTIPLE NEURITIS SUBSEQUENT TO THE GRIPPE.

Severe attacks of neuralgia accompany or follow the grippe in about 25 per cent. of the cases, and are an evidence of direct action of the poison upon the nerve trunks. Thus trigeminal neuralgia, occipital neuralgia, intercostal neuralgia, and sciatica frequently develop and sometimes run a very severe and long course. They are occasionally bilateral and symmetrical, which is rare under other conditions.

Many cases of local neuritis appearing in one or in several nerves in the body at once have been observed after the grippe. Any of the cranial or spinal nerves may be involved.

It is not uncommon for such a neuritis to attack symmetrical nerves on both sides of the body. Thus I have seen both peroneii affected, both ulnar nerves affected, both median nerves affected, and the brachial plexus affected on both sides. In all these cases the neuritis ran a typical course.

Draper,[2] Remak,[3] and Bernhardt[4] recorded cases of multiple neuritis accompanied by acute ataxia following the first epidemic of influenza, 1890. Mills,[5] Buzzard,[6] Eisenloch,[7] Savage,[8] and others subsequently reported cases of multiple neuritis with sensory symptoms and with paralysis without ataxia, and various writers, in confirming these observations, have described cases in which the cranial nerves of one or both sides, the ocular nerves, the facial nerves of one or both sides, and the palate have been involved. In a few cases œdema in the limbs has been noticed as a prominent symptom. These cases have developed within one or two weeks of the attack of influenza, have reached their height about one month after the attack, and have remained for many months. A few cases with rapid recovery within two months have been seen. There are no particular features to distinguish these cases from the ordinary types of neuritis, and there is no special treatment for them.

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[1] Kast. Deut. Arch. f. klin. Med., 1886, Bd. xl. S. 41.
[2] New York Medical Record, 1890, p. 239.
[3] Berliner klin. Wochen., 1890, p. 181.
[4] Ibid., p. 643.
[5] Boston Medical and Surgical Journal, 1892, p. 349, 405.
[6] Transactions of the Philadelphia Medical Society, 1892.
[7] Neurolog. Centralbl., 1893, p. 380.
[8] Journal of the American Medical Association, July 24, 1897.

MULTIPLE NEURITIS SUBSEQUENT TO VARIOUS INFECTIONS.

It is still somewhat a matter of conjecture whether cases of paralysis following typhoid, typhus, and malarial fevers are due to an affection of the peripheral nerves or of the spinal cord. The researches of Pitres and Vaillard[1] have shown that extensive degeneration of peripheral nerves is to be found in the bodies of patients who have died of typhoid fever, and they have also demonstrated that these fevers are often followed by local neuritis as a sequel. Cases of multiple neuritis with autopsies have not as yet been reported after typhoid or typhus. It is so common in severe cases of these diseases to use alcoholic stimulants as a remedy that it is not impossible that in some of these cases the real cause of the disease was alcoholic poisoning. In several hospital cases which I have seen this has been undoubtedly the cause of the supposed post-typhoid neuritis, the symptoms being identical with those occurring in alcoholic cases and including cerebral symptoms, delusions, and loss of memory. Hence great care should be taken in reaching a conclusion that any post-typhoid case is really due to the infection, although all writers on typhoid emphasize the possibility of multiple neuritis as a sequel.

Buzzard recorded[2] two cases of paralysis following malarial fever, in which all the symptoms pointed to an affection of the peripheral nerves. In this country Gibney has described several cases of paralysis of the extremities, of sudden onset, rapid course, and prompt recovery under large doses of quinine, which he considered malarial. The following case, seen with Holt, was of this nature:

A healthy boy, aged ten years, was suddenly seized with a chill and fever and with pains of a severe kind in both legs, associated with weakness so that he could hardly stand. The pain was referred to the sciatic nerves and was accompanied by numbness and tingling of the feet and legs. There was marked tenderness both in the course of the sciatics and in the muscles of the thigh and leg. There was loss of tendon reflexes, preservation of skin reflexes, and marked paresis in all the muscles of the legs, so that he required help in walking. There was no ataxia and no incontinence of urine. The symptoms lasted about twenty-four hours and then decreased in severity during the following day, but recurred on the third, and, after a remission, on the fifth day, with lessened severity. The periodicity of the affection and the rapid cure under quinine left no doubt as to its malarial nature. During the third day, at the time when I saw him, there was redness along the course of the sciatics and in the region of the right median nerves and extreme tenderness along these nerve trunks, in addition to the symptoms mentioned. The electric condition was, unfortunately, not tested; but in Westphal's case faradic contractility was totally abolished during the attack.[1] In this case the active manifestation of the malarial poisoning was by general neuritis. In Buzzard's cases the malarial attacks preceded the nervous affection. The tenderness in muscles and nerves left no doubt regarding the peripheral nature of the disease.

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[1] Rev. de Méd., 1885, p. 980, Des Névrites peripheriques.

[2] Paralysis from Peripheral Neuritis, p. 104, London, 1886.

I have recently seen a very severe case of general neuritis with total paralysis in both arms and legs lasting many months, and paralysis of the diaphragm which lasted for two weeks and was attended by rapid pulse and great exhaustion. In this case the only cause ascertainable was an attack of severe dengue fever which immediately preceded the paralysis. The patient was an engineer and contracted the disease in South America and was brought to New York with great difficulty. He had cerebral symptoms for some weeks at the height of the disease.

Neuritis following variola is a rare complication, and the following case is the only one on record in which an autopsy showed the lesion to be a multiple neuritis.

A young man had varioloid in November, 1881, and while convalescing, six weeks later, began to suffer from severe pains in his four extremities, especially in the joints of his arms, which were diagnosticated as rheumatic, although there was no fever. Soon after there followed a true paresis, with progressive atrophy of the muscles of the forearms and legs. The muscles at the same time became very tender to touch or pressure. The tendon reflexes were much diminished. Reaction of degeneration developed in all the paretic muscles. The pains in the joints and limbs continued, but were less severe than at the outset. The sensibility of the skin was about normal. The nerve trunks were tender to pressure. Profuse, offensive perspiration in all four extremities was a distressing symptom. There was no tendency to bed-sores, but an extensive pemphigus developed in the legs, and then the pains became more severe. The patient died of pneumonia.

Autopsy showed the brain œdematous and the cord in a state of hypostatic congestion. The pathological changes of importance were found in the nerves and muscles. The majority of the nerves of all the extremities were found in a state of degeneration and atrophy. There was marked degenerative atrophy and fatty degeneration of the muscles.[2]

Scarlet fever,[3] measles,[4] whooping-cough,[5] and mumps have been known to be followed by neuritis, both local and multiple, but the cases are so rare as to require only a mention.

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[1] Neurolog. Centralbl., 1885, p. 187.

[2] P. Grocco, Milano. Centralbl. f. med. Wissen., 1885, p. 693.

[3] M. J. Bassette. Journal of Nervous and Mental Diseases, 1892, p. 461.

[4] Allyn. Medical News, 1891, p. 617.

[5] Leroux. Allg. Wiener med. Zeitsch., 1898, Nr. 29.

Multiple neuritis following erysipelas or of septicæmic origin has been reported by French[1] and German[2] authorities. I have seen a severe case in which the only cause found was a long-continued suppurating tooth. This patient had a continued fever during the early part of the disease. Bury[3] reported a case in which an infected wound of the finger gave rise to secondary abscesses, and then multiple neuritis developed.

Both local and multiple neuritis have been known to develop subsequent to attacks of gonorrhœa. They have no special characteristics.

German[4] authors have described many cases of so-called puerperal multiple neuritis; cases that appeared to be of the infectious type and due to septicæmia following abortion, miscarriage, or delivery. These cases are not to be confounded with the local neuritis of the sacral plexus consequent upon compression of this plexus during labor, nor are they to be confounded with cases of myelitis consequent upon anæmia of the spinal cord subsequent to confinement. The cases recorded by the Germans have been of general widespread multiple neuritis of the septic type. Such cases have not been observed in this country, and in the large material at the Sloane Maternity Hospital there is no record of multiple neuritis occurring in the course of pregnancy or after labor. Cases of neuritis developing during pregnancy and traced to the exhaustion following long-continued vomiting are not unknown,[5] but are not of the septic type.

LEPROUS NEURITIS.

Multiple neuritis occurring in leprosy is a very rare affection in this country, but is of some interest on account of the fact that it is the only form of neuritis in which the bacillus of any disease has been found in nerves. This bacillus locates itself entirely in the interstitial tissue of the nerve trunks within the sheath, in the perineurium, and by its direct irritation produces hyperplasia of connective tissue. This causes a compression of the nerve fibres and a secondary degeneration in them. Here and there along the course of an affected nerve are found small bulbous enlargements where the connective tissue is particularly thick. The changes in the nerves themselves are those of simple degeneration. The lesion is due to the direct effect of the bacilli and is not secondary to any infection or any poisoning conveyed to the nerves through the blood. For this reason it is a localized neuritis, but is multiple in its character, inasmuch as very many of the terminal branches of the filaments of the nerves are involved. (See Plate X.)

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[1] Charcot. Revue neurologique, 1893, Nr. 1 et 2.

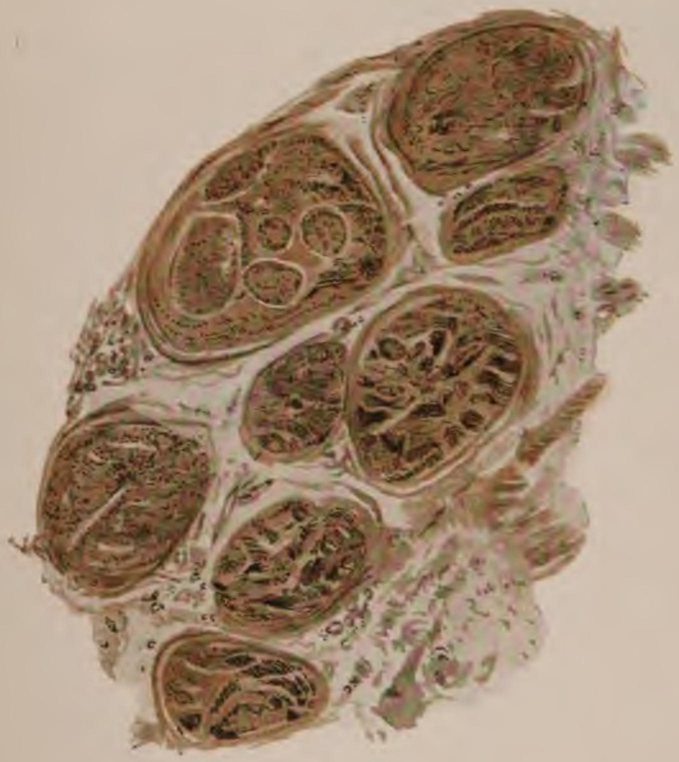
[2] Gerhardt. Deutsche med. Wochen., 1898, p. 14. Kraus, Wiener klin. Wochen., 1897, Nr. 40.

[3] Ross and Bury. Peripheral Neuritis, p. 291, 1893.

[4] Moebius. Münchener med. Wochen., 1890, Nr. 14. Mader, Wiener klin. Wochen., 1895, p. 537. Jolly. Arch. f. Psych., 1897, p. 650.

[5] Stiefel. New York Polyclinic, 1893, March. Stembo, Deutsche med. Wochen., 1895, p. 461.

PLATE X.



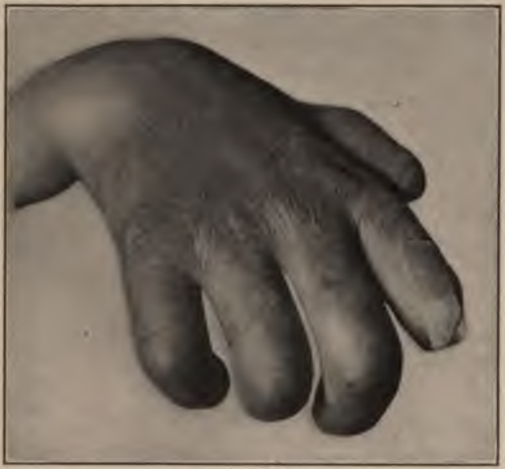
Cross-section of Sciatic Nerve in a Case of Leprous Neuritis. (Marchi stain.)

The degenerated nerve fibres in the various bundles of nerves are stained black. The infiltration of the perineurium and epineurium with granulation cells, not shown by this stain, has forced the bundles apart.

(Flatau, Spec. Pathol. u. Therap., Nothnagel, Bd. xi., Taf. viii.)

Symptoms. The symptoms of leprous neuritis are the gradual development of irregular areas of anæsthesia, the occurrence of paralysis and atrophy in the peripheral parts of the limbs, and the production of trophic disorders. The anæsthesia is extremely irregular in its distribution, depending wholly upon the branches of the nerves which are affected by the disease, irregular plaques of anæsthesia appearing here and there upon the hands and feet or upon the forearms and legs, and on the trunk, and not infrequently upon the face. These anæsthetic areas do not, as a rule, correspond to the distribution of any one nerve, nor are they symmetrical, as in ordinary cases of multiple neuritis; but are extremely irregular. They are attended by numbness and tingling and burning sensations, or a pain of slight degree. The loss of sensibility may affect touch, temperature, and painful sensations equally, or any one of these may be affected alone. A loss of pain and temperature senses, while touch is preserved, is not uncommon, hence the disease has been mistaken for syringomyelia. The paralysis is usually found in the very small muscles of the hands and feet, but may extend to the longer muscles of the extremities, and not uncommonly a few of the facial muscles are paralyzed. A progressive atrophy occurs in the paralyzed muscles, with reaction of degeneration. There is a loss of mechanical excitability in the muscles affected. The trophic disturbances present in the disease, viz., whitish colored plaques on the skin, peculiar thickening or atrophy of the skin, the glossy surface, the ulcerations, and the gradual loss of substance—are to be regarded rather as a part of the disease leprosy than as evidence of the local affection of the nerves.

FIG. 31.



Atrophy and trophic changes in hand and fingers in a case of leprous neuritis.

Fig. 31 illustrates the condition occasionally resulting from an extreme condition of anæsthetic leprosy, the photograph being taken from a patient seen by me in consultation with Piffard—one of the few cases of the kind seen in New York within the past twenty years. The ends of the thumb and forefinger had been eroded.

The disease is a slowly progressive one and treatment appears to be of no avail.

The differential diagnosis between lepra anæsthetica and syringomyelia is sometimes somewhat difficult, as the trophic disturbances, anæsthetic areas, and muscular palsies of irregular distribution occur in both diseases; but the distribution of the anæsthesia is usually greater in syringomyelia and begins in (if it is not entirely limited to) the upper extremities. White plaques are not common upon the skin in syringomyelia. Bulbous swellings upon a nerve trunk are not felt in syringomyelia, while in lepra anæsthetica there is an absence of those symptoms of spastic paraplegia, disturbance of the bladder and rectum, and increased reflex activity of the legs, which are commonly found in syringomyelia.

References may be made to the articles of Schultze,[1] Dehio,[2] Zambacho,[3] Laehr,[4] and Blaschko.[5]

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[1] Deutsche. Arch. f. klin. Med., 1888, Bd. 43.

[2] St. Petersburg Wochen., 1889, No. 42, and 1890, No. 48.

[3] Verhandlungen des internationale wissenschaftlichen Lepra Conferenzes zu Berlin, October, 1897.

[4] Arch. f. Psych., Bd. xxx. 323.

[5] Die Nervenkrankheitserscheinungen der Lepra, Berlin, 1899.

CHAPTER VIII.

EPIDEMIC MULTIPLE NEURITIS.

BERIBERI OR KAKKE.

In 1882 Professor Scheube, of Tokio, Japan, called the attention of European physicians to the existence of a peculiar affection prevailing among the Japanese.[1] It was called kakke, from the two Chinese words—kiaku, meaning legs, and ke, meaning disease. It had been known among the Chinese for centuries, being mentioned by name in Chinese medical books written 200 B.C., and fully discussed by an eminent author in 640 A.D. It ceased, however, to prevail in China about two hundred years ago, and of late it is found chiefly in Japan. There its importance is considerable, since it is so prevalent that in 1877, 14 per cent and in 1878, 38 per cent. of the men serving in the army suffered from it. It is considered a miasmatic infectious disease by Scheube, although an eminent Japanese authority considers it due in some way to the diet of rice. That diet has something to do with its occurrence is proven by the fact, communicated to me by Dr. Wallace Taylor, long a resident of Osaka, that since wheat has been substituted for rice in the diet of some of the barracks and prisons in Japan the disease has been less common. It occurs in epidemics, but is always endemic in Japan. It rarely attacks Europeans. While not contagious, the disease is transmitted by human beings, for outbreaks have occurred on islands formerly free after the landing of persons who were affected. It affects females rarely, only 9 per cent. of the cases being in women; and it is the youth of the land, between the ages of sixteen and twenty-five years, who suffer. Exposure to damp and cold in crowded dwellings, such as barracks, increases the liability to the affection. The majority of the cases occur during the hot, damp months, but some are always under observation.

This disease is not, however, confined to Japan. It has been observed for many years in the islands of the Pacific Ocean, especially in the Philippines, in India, Ceylon, on the west coast of the Red Sea, in Borneo and New Guinea, in Brazil and Cuba, on the isthmus of Panama, and in the Dutch possessions in the China Sea.[2] It is there known under the name of beriberi. It is endemic in these regions, but occasionally occurs as an epidemic. An interesting

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[1] Deutsche Arch. f. klin. Med., vols. xxvii. and xxxi.

[2] B. Scheube. Diseases of Warm Countries, p. 190, London, 1903, where a complete literature is to be found.

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account of such an epidemic, occurring in 1882–83 in Manila, in the Philippine Islands, has been given by Koeniger.[1] It appears that in the fall of 1882 an epidemic of cholera occurred in Manila of such severity that 20,000 persons, in a population of 400,000, were affected. As a precaution against this disease the native population lived for several months exclusively upon rice, refusing to eat fruit or fish, which are their other chief articles of diet. As the epidemic was subsiding a terrible cyclone devastated the city, destroying the light wooden houses, and leaving 60,000 families homeless; and these poor people were exposed for several weeks to the inclemency of the weather, which at this time of the year is rainy. A few days after the cyclone the epidemic of beriberi began, and, as the disease had never before appeared in Manila, the unknown affection excited great alarm. This was increased by its fearful mortality, 60 per cent. of the early cases proving fatal. Europeans were exempt, with two exceptions, and the Chinese population did not suffer greatly, but among the natives the epidemic was widespread. Thus, in one suburb of Manila, of 25,000 inhabitants, 300 died in the course of eight weeks. Men and women were equally affected and persons of all ages, except young children, were attacked. The disease terminated fatally in from ten days to five weeks after its onset; but as time went on the proportion of recoveries increased, and by the end of March, 1883, it had almost disappeared. The months from October to March are the dry, cool season in the Philippine Islands, although the climate is tropical. Exposure to heat could hardly be considered a cause of this epidemic, but whether the exposure to cold and damp or the diet of rice or the transportation of some infectious agent by the cyclone was the cause could not be determined.

Sporadic cases of beriberi, or kakke, occasionally appear in our hospitals, usually in the persons of Chinese or Malay sailors, or in the persons of travellers from tropical climates who have been exposed to the infection in the place from which they came.[2] In 1881 a Brazilian naval vessel entered San Francisco with a large number of the crew affected by the disease. They were sent to the United States Marine Hospital and attended by Dr. Hebersmith, who gave an interesting account of the circumstances leading to the development of the disease in the *United States Marine Hospital Report* for 1881.

In 1886 a commission was appointed by the Dutch Government to investigate the subject of its nature, and the published report contains the following statements:[3] The disease is caused by a microorganism resembling the bacillus of splenic fever, though somewhat smaller, which colors with fuchsin and gentian-violet, and can be seen with a power of 560 diameters. These bacilli are found in the blood, lungs, heart, brain, cord, and nerves of the patients, and can be cultivated outside of the body. The germs infect wooden dwellings chiefly. They may be conveyed by articles of clothing, and probably enter the body by the lungs. Direct contagion has not been observed. A potent predisposing cause to their reception in the body and to the development of the disease is lack of nutrition, consequent upon exposure to damp and cold, and upon insufficient or bad food. It must, however, be added that a most thorough examination in a recent case by Prudden failed to reveal the presence of such bacilli, and Scheube in his recent book states that the virus is unknown. In the light of these investigations a new view is taken of an epidemic of a peculiar kind which occurred in France in 1828. Buzzard has found an account of this, prepared by Graves, in which the symptoms are so fully detailed as to leave no doubt that it was an epidemic of multiple neuritis.[1]

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[1] Deut. Arch. f. klin. Med., xxxiv.

[2] Seguin. Medical News, Dec. 11, 1886. Bondurant, Jour. Nerv. and Ment. Dis., Dec., 1900.

[3] Deutsche med. Wochen., December 9, 1886. See also Harada, Neurolog. Centralbl., 1885, p. 326.

Symptoms. The cases of beriberi are divided into two general classes, according to their severity:

There are, first, slight cases, in which the onset is gradual, being usually preceded by a little fever, coryza, and conjunctivitis, which cease when the actual symptoms commence. The patient first notices a weak and heavy feeling in the legs, and finds that he tires so easily that he cannot walk as much as usual. The fatigue is soon associated with numbness and pain in the legs, and with a slight œdematous swelling. Then, if not before, palpitation of the heart, oppression and weight in the epigastrium, loss of appetite, and general malaise are felt, and the patient finds it necessary to apply for treatment. An examination then shows some diminution of power in the feet and legs and also in the hands, with loss of tendon reflexes and much tenderness in the muscles, which show a diminished electric excitability. There is never any ataxia, though the patient sways when his eyes are closed. There is discovered a slight degree of anæsthesia of irregular distribution, chiefly in the legs and in the radial nerve region on the forearms. Though the patients look pale, examination of the blood does not show any marked anæmia.

The circulation in the extremities is sluggish. The heart is irregular and rather rapid, and the œdema of the extremities indicates a failure of its power. Wallace Taylor[2] found that a sphygmographic tracing is characterized by a sudden upstroke in ventricular systole, by a precipitous descent from the apex of the percussion wave, and by dicrotism. Beyond this point these cases, which make up the majority, do not advance. They usually recover in a few days, or at most a month, although a few become chronic and require several months before the cure is complete.

There are, secondly, severe cases. These may present three different types. There is, first, the atrophic or dry type, in which, after an onset similar in nature to that in the slight cases, but much more rapid, the weakness develops into a true paralysis associated with marked wasting of the muscles and reaction of degeneration, with great diminution of galvanic excitability. Within a week the patient has to go to bed, and then the paralysis soon spreads from the legs to the arms, and may involve the trunk and even the face. The entire muscular system wastes away until the patient is a mere skeleton. In addition to the motor symptoms there is great sensory disturbance. The suffering from pain, paræsthesiæ, and general muscular tenderness is extreme, and the patient lies totally helpless and unable to tolerate the lightest touch. The skin may be glossy. There is usually some anæsthesia, but it is never complete, and it does not involve the trunk. The temperature sense is seldom affected. Pain may be delayed in transmission. There are no gastric symptoms and no œdema. Some cases prove fatal from general exhaustion or intercurrent disease, but the majority recover after a convalescence which lasts a year or more, during which the muscular system is rebuilt.

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[1] On Paralysis from Peripheral Neuritis, p. 37.

[2] Studies in Japanese Kakke, by Wallace Taylor, M.D. Osaka, 1886.

There is, secondly, the hydropic or wet type. In these heart failure appears early and is associated with a marked decrease of arterial tension and much œdema of the entire body, the effusion into the cavities being added to that beneath the integument. The swelling of the œdematous parts conceals the atrophy which is going on in the muscles, but this is indicated by the paralysis, which is as severe as in the preceding form, and it becomes evident during recovery when the œdema has subsided.

There is, thirdly, the acute pernicious type. In this all the symptoms of the two former types appear in rapid succession, and, in addition, gastro-intestinal symptoms and a suppression of urine combine to make the condition an alarming one. Effusions into the pleura and pericardium appear early. The pulse becomes small and irregular, and cyanosis indicates the heart failure which precedes death. There are no constant changes in the blood, and leucocytosis has not been found.

In this form the disease may run its course in two weeks to a fatal termination. This was the form which chiefly prevailed in Manila, the cases of the atrophic form being the ones which recovered.

The severity differs much in different epidemics, the mortality varying from 2 per cent. in Japan to 60 per cent. in Manila. It is usually not above 3 per cent. In all the forms there is some danger of a sudden heart failure, and this is usually the cause of death.

Treatment. As to its treatment, it may be mentioned that quinine fails to influence its course and that heart stimulants to combat the dangerous complications, hypnotics to counteract the pain and insomnia, and general tonic treatment have proved of the greatest service. Change of climate is often attended by recovery. In the stage of recovery electricity and massage have been employed with advantage.

CHAPTER IX.

MULTIPLE NEURITIS ASSOCIATED WITH OTHER DISEASED STATES OF THE BODY.

Multiple Neuritis and Rheumatism, Gout, Diabetes, Tuberculosis, Syphilis, and Carcinoma. Multiple Neuritis of Unknown Origin. Senile Polyneuritis.

THE occurrence of multiple neuritis in connection with certain other diseases has been observed in so many cases as to have led to the hypothesis that there must be some causal relation between the two conditions. The diseases in which neuritis develops are of a nature to affect the general health, to impair the nutrition, and to produce a constitutional disturbance; but it becomes evident, from the fact that only a small percentage of the patients suffering from these diseases develop neuritis that some other cause must be active in conjunction with the disease. A congenital or acquired weakness of the nervous system is probably present in certain individuals and predisposes them to this complication, when they fall victims to other affections. The diseases with which neuritis is frequently associated are rheumatism and gout, diabetes, tuberculosis, syphilis, carcinoma, senility, and arterial sclerosis.

MULTIPLE NEURITIS AND RHEUMATISM.

Multiple neuritis is very rarely to be traced to rheumatism. The so-called "rheumatic" cases of the Germans are cases due to exposure to cold and do not present the characteristic symptoms of rheumatic fever and joint affections. In many cases of neuritis some pain in the joints on motion may be present, but it is very seldom that the red, swollen, œdematous, hot joints of rheumatism are found. A critical review of the cases of multiple neuritis supposed to be rheumatic[1] shows no conclusive evidence of the coexistence of the two diseases. It is not enough to consider pain and tenderness about the rheumatic joint as evidence of neuritis when anæsthesia and paralysis in the domain of these tender nerves fail to appear. Nor is it sufficient evidence of rheumatism to find painful joints in cases of neuritis without the constitutional effects of the disease. Hence it seems very questionable whether we are justified in considering rheumatism as a cause of multiple neuritis. It is admitted that local neuritis is not infrequently produced in the vicinity of a rheumatic joint, as in the cases fully described by Bury.[1] Thus, in rheumatism of the elbow, the ulnar nerve may become inflamed, swollen, tender to pressure, and paralysis and atrophy of the interossei and thenar eminences may ensue, with anæsthesia in the hand. So, too, peroneal neuritis may result from a rheumatism of the knee. I have seen several cases of this kind. In such cases there is a localized neuritis due to extension of the inflammatory processes directly from the joint to the nerves passing over it. This cannot be considered, however, as a neuritis due to the state of the blood which produces the rheumatism. Furthermore, an error is often made when the tender, stiff joints which develop late in the course of neuritis are termed rheumatic. In the later stages of alcoholic neuritis, when the tenderness of the skin and muscles and the pain produced by any motion have led the patient to keep the hands and fingers and toes perfectly fixed and immovable for weeks, an ankylosis of the smaller joints often develops, and this may be accompanied by some thickening of the articular surfaces and is often attended by thickening of the skin over the knuckles, and by profuse sweating of the hands and feet. This may give rise to a condition not unlike that resulting from chronic rheumatism. It is not, however, rheumatic, but is the natural sequel of inactivity in a joint which is fixed, and is a trophic symptom of neuritis.

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[1] *E. g.*, the cases cited by Steiner. Deut. Arch. f. klin. Med., lviii. p. 240.

It is evident, therefore, that it is a mistake to regard neuritis as a frequent result of rheumatism, or to establish any causal relation between the two diseases. In case the two diseases coincide or follow one another, each should be treated separately.

In the course of a case of chronic articular rheumatism or of chronic arthritis deformans, multiple neuritis may develop from any cause. I have seen several such cases; but here, again, there is never any certain evidence of a causal relation between the two affections.

MULTIPLE NEURITIS FOLLOWING GOUT.

Neuralgia and localized neuritis in single nerves have been frequently observed as a complication of gout. Of late, English writers have called attention to the frequency of attacks of multiple neuritis affecting symmetrical nerves on both sides of the body as an occasional sequel or accompaniment of this constitutional affection. Sciatica and brachial neuritis are frequently traced to this cause. Buzzard,[2] in particular, has described several cases in which a sudden attack of pain and numbness in the hands has been followed by weakness and loss of faradic excitability in the muscles of the hands and forearms, and in one case this condition soon appeared in the feet. These patients were gouty persons and acute attacks of gout had preceded or accompanied the nervous symptoms. Ebstein[1] and Grube[2] and Remak[3] have published similar cases, and all agree that a direct relationship between gout and neuritis is proven. A general peripheral neuritis, such as the form occurring after alcoholic poisoning or after exposure to cold, has not been observed. For this reason the following case, in which both peroneal and anterior tibial nerves were affected and the smaller branches of the nerves in the hands were also involved, may be recorded as of interest:

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[1] Medical Chronicle, June, 1888.

[2] Buzzard. Harveian Lectures, 1885, Lancet, vol. ii. p. 983. Dyce Duckworth, Gout. London, 1893, p. 247.

The disease began with a severe itching and burning sensation about the toe-nails and upon the dorsum of the right foot, which soon developed in the left foot also. This itching and burning sensation then extended up both legs to the knee, but was not at first attended by any change in the appearance of the skin, or by actual anæsthesia. After several remissions the symptoms became more intense, and the burning and itching were accompanied by the appearance of a crop of minute vesicles. The feet then became swollen, the skin became glossy, red, and extremely tender to the slightest touch, and the sensation was one of great pain throughout both lower extremities. Any moist dressing upon the surface intensified the pain, but dry dressings or oily applications seemed to give some relief. Large serous blebs formed upon the soles of the feet and about the toes, and there was considerable eczematous exudation and a scaly appearance of the skin of the entire legs. After this condition had lasted for three months similar itching, and burning began in the hands about the fingers with very marked, deep-seated paræsthesia. Fine vesicular pin-point elevations in the skin of the hands also appeared, but these did not go on to the formation of blebs, as in the feet. After two months the skin of the legs had become thickened, cracked, and shiny with crusts all over the surface, and when these had peeled off the surface was red and very sensitive to light pressure. The nails of the toes were thickened, rough, striated, and black. It was said that they had not grown at all during six months. Tactile sensibility was diminished over both feet and both legs, but any touch was attended by great pain. The muscles of the feet and legs had become extremely atrophied and were almost entirely paralyzed; no electrical examination could be made on account of the extreme sensitiveness. At this time sciatica was complained of as high as to the buttocks. The knee-jerks, at first exaggerated, were subsequently lost.

When I saw the patient, a woman of about fifty years of age, seven months after the onset, she was unable to stand or bear her feet upon the floor, but the paralysis had subsided and she could move her ankles; but could not move her toes. There was very marked tenderness to touch over both legs and upon both feet, and the tenderness of the nerves upon the soles of the feet was extreme. The skin of the feet was dry, scaly, and cracked. The nails were badly discolored, ridged, and dark and rough, excepting near the matrix, where a new growth of nail, about one-half inch in depth, had begun. Any covering applied to the feet caused intense burning and itching, so that she kept the feet entirely uncovered both by day and night. There was nothing in the way of local treatment that relieved this itching and burning, though every form of local application had been tried. The ball of the foot was red, the rest of the foot white; but during an attack of pain the feet became scarlet, or sometimes became very much more pallid than usual. It was evident that the condition in the feet was that of erythromelalgia.

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[1] Ebstein. Deutsche med. Wochen., 1898, p. 489.
[2] Grube. Münchener med. Wochen., 1899, p. 23
[3] Remak. Nothnagel's spec. Path. u. Ther.,. vol. xi., Th. iii., p. 623.

There was a similar burning sensation and itching felt in the hands from time to time, but there was no apparent malnutrition of the skin.

The patient came of a very gouty family and had many indications of the presence of gout, and all these symptoms gradually subsided in the course of a year under treatment directed exclusively to the gouty state, namely, dietetic treatment and alkalies given freely. The coal-tar products appeared to give some little relief to the local symptoms. I have seen the patient several times in the past five years and there has been no return of the affection.

I have seen several other cases that were quite similar to this in their symptoms and course and have no doubt that they can be classed together as gouty neuritis.

MULTIPLE NEURITIS FOLLOWING DIABETES.

Neuralgia in the course of diabetes is a very common occurrence, and probably is an indication that the condition of the blood in this disease produces an imperfect nutrition of the nerves. The loss of knee-jerk often observed in this disease (30 per cent. of cases) supports this hypothesis.[1] The sciatic nerve seems to be the one more commonly affected than any other nerve.[2] I have seen many cases, and think it important to look for sugar in the urine in every case of sciatica. Bruns has called attention to the frequency of crural and obturator neuralgia and neuritis in the course of diabetes, and Buzzard[3] has observed several cases of brachial plexus neuritis following diabetes. In some cases the attacks of neuralgia have been bilateral.[4] In some cases they have gone on to neuritis. The nervous symptoms occurring in the course of diabetes may be not only neuralgic pains of a sharp, shooting character, but also various forms of paræsthesia, numbness, and burning sensations. Patients often complain of severe cramps in the legs, especially at night, and these may precede or may accompany sciatica.

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[1] Ziemssen. Münchener med. Wochen., 1885, p. 618.
[2] Auerbach. Deutsches Arch. f. klin. Med., 1887, p. 484.
[3] Buzzard. British Medical Journal, 1890, vol. i. p. 1421.
[4] Williamson. Medical Chronicle, 1892, November; Lancet, 1897, vol. ii. p. 138.

Multiple symmetrical neuritis of extensive distribution, not unlike that occurring after poisoning by alcohol, both of the paralytic and of the ataxic types, has been ascribed to diabetes, but is exceptional.[1] Several authors have described cases of so-called neurotabes peripherica diabetica, which have resembled locomotor ataxia, but have gone on to recovery.[2] In some of these cases the diagnosis from true tabes was difficult, especially as perforating ulcer of the foot occurred.[3] The tendency to gangrene in diabetes is not traceable to neuritis. The intensity of the neuritis, according to Gowers, is not related to the amount of the sugar in the urine.

The prognosis is good in all these forms of neuritis, as they recover when the sugar disappears from the urine.

The treatment will necessarily be directed to the original disease as well as to the complication.

MULTIPLE NEURITIS AND TUBERCULOSIS.

Cases of multiple neuritis occurring in tuberculous patients require some consideration. If a review of cases of multiple neuritis with autopsy be made it will be found that quite a number of the patients died of phthisis. There is little evidence to prove that the neuritis was due in these cases to the tuberculous disease. No one has found tubercles in the nerve trunks in these cases, and bacteriological examination has not demonstrated the presence of bacilli in the nerves. For this reason it is not possible to speak of a tuberculous multiple neuritis. There is no doubt, however, that the existence of a grave constitutional affection may lead to such a general impairment of nutrition in the nerves, as well as in other organs, as to predispose such a patient to an attack of neuritis. Hence especial mention must be made of neuritis as a complication of phthisis. It should not be forgotten, however, that in phthisical patients who have used alcohol freely the neuritis may owe its origin to this poison. Oppenheim[4] has reported cases of multiple neuritis in tuberculous patients which went on to recovery. The following case died:

A young girl who was suffering from phthisis took a severe cold and began to have pains in the joints, weakness, and numbness of the lower extremities. The weakness of the legs increased rapidly and was attended by rapid, diffuse atrophy. The same symptoms soon developed in the arms. Four weeks after the onset partial reaction of degeneration was present in all the extremities, and a total paralysis of the legs and nearly complete paralysis of the arms were present. In the course of the disease, which lasted five months, there was little pain, but great tenderness of the muscles and hyperæsthesia of the skin. The sensation was diminished in the feet and hands, but this was slight in comparison with the motor symptoms. The paralysis finally attacked the trunk muscles and the diaphragm, while the beginning of rapid heart action was thought to indicate paralysis of the pneumogastric nerve. Great prostration, delirium, incontinence, bed-sores, and œdema of extremities, with fever, preceded death.

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[1] Pryce. Brain, 1893, vol. xvi. p. 416.

[2] Leichtentrett. Inaug. Dis., Berlin, 1893.

[3] Raymond. Leçons sur les malad. du syst. nerv., 1895, vol. ii. p. 331.

[4] Zeitsch. f. klin. Med., 1886, p. 230.

A high degree of degeneration of the peripheral nerves, including the phrenic and pneumogastric, was found at the autopsy, with a moderate degree of atrophy of the muscles. Spinal cord and anterior motor roots were normal. The changes in the nerves were similar to those after section, viz., a simple degeneration of the fibres with some increase in the connective tissue, but there were no evidences of changes in the vessel walls, no infiltration with cells. The nerves contained very few normal fibres. The myelin sheaths were found in all stages of destruction even to an entire absence of contents of the sheath of Schwann. Throughout the fibres fatty and granular masses were found. The sheath of Schwann did not, however, show an increase of nuclei.[1]

Such a case as this would have been ascribed, a few years ago, to a tuberculous spinal meningitis. It is not my purpose to dispute in any way the fact that many cases of paralysis occurring in the course of phthisis are due to lesions of the central nervous system and its membranes, but I desire to emphasize the fact that a multiple neuritis may produce paralysis in tuberculous individuals, and the importance of appreciating this possibility cannot be too strongly urged, since the treatment, as well as the prognosis, will differ widely, according to the diagnosis made.

It has been known for some time that local neuritis may complicate phthisis, and the researches of Pitres and Vaillard have confirmed this fact. They have described (*a*) a latent neuritis in which lesions in the nerves were found post-mortem, but no symptoms had appeared; (*b*) neuritis causing paralysis and atrophy in the muscles, and (*c*) neuritis with sensory symptoms only. They are inclined to ascribe many of the nervous disturbances arising in the course of consumption to an affection of the peripheral nerves; and other writers have confirmed these views and have recorded cases in which all forms of cranial and spinal neuritis have appeared. But here, again, the neuritis must not be termed tuberculous, as it has nothing characteristic of that affection in its pathology.

MULTIPLE NEURITIS AND SYPHILIS.

There has been much discussion in regard to the existence of a syphilitic multiple neuritis. The admitted frequency of syphilitic exudations in nerve trunks, especially in the cranial nerves and about the roots of the spinal nerves, and the admitted existence of gummy growths in the plexuses and in the nerves, and of syphilitic endarteritis in the vessels accompanying the nerves have made it seem not improbable that a true syphilitic multiple neuritis might occur. The prevalence of syphilis has resulted in the development of multiple neuritis in many patients who were syphilitic, and this has also awakened the suspicion that syphilis might be the cause of the neuritis. The syphilitic poison has been designated by some authors as the cause in these cases; by others the effects have been ascribed to the post-syphilitic toxin which seems to play such a prominent part in the production of tabes. Cases have been reported in all stages of the disease. It is only in a few of the reported cases, however, that a true syphilitic multiple neuritis can be admitted. In the cases of Fordyce,[1] Spellmann and Etienne,[2] and Fry,[3] the development of multiple neuritis in the course of the second stage of syphilis, the accompanying numerous syphilitic deposits in the skin and periosteum, and the rapid improvement under antisyphilitic treatment give a certain probability to the assertion that these cases were of specific origin. It must be acknowledged, however, that such cases are extremely rare. At a discussion at the New York Neurological Society it was found that no one of the members had seen a case of multiple neuritis undoubtedly syphilitic, and a review of the literature has convinced me that many of the cases reported as such were not actually due to the disease.

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[1] Vierordt. Arch. f. Psych., 1883, vol. xiv. p. 3.

MULTIPLE NEURITIS AND CANCER.

Auche[4] collected from the literature and published a number of cases of neuritis developing in the course of carcinoma, especially of the stomach. Some of these were local neuritis due to a direct extension of the carcinoma into adjacent nerve trunks. Others were general multiple neuritis in the peripheral branches. The lesions were found post-mortem to be of the type of degeneration in the finer nerve filaments, not unlike those found by Pitres and Vaillard in tuberculous patients. In some of these cases no symptoms had appeared during life. In others minor sensory symptoms were noticed: paræsthesiæ, and anæsthesiæ, and pain. In a few cases the symptoms of a general multiple neuritis, typical sensory and motor paralysis developed. This was seen in cases of Miura and Remak,[5] where the autopsy confirmed the diagnosis. In this connection it is to be remembered that processes of degeneration are going on continually in normal nerves in a state of health, that these processes are undoubtedly intensified and regeneration is delayed in conditions of wasting disease, such as tuberculosis and carcinoma, and also in senile conditions or where the circulation and nutrition are interfered with, as in states of extensive arterial sclerosis. Hence, it is not proper to lay too much stress upon lesions of this kind found after death when no symptoms have appeared during life. And while "latent neuritis" may awaken interest as a pathological curiosity, it is of no importance to the clinician.

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[1] Boston Medical and Surgical Journal, 1890, p. 39.
[2] Revue neurologique, 1897, p. 28.
[3] Journal of Nervous and Mental Disease, 1898, p. 594.
[4] Revue de méd., October, 1890.
[5] Berliner klin. Wochen., 1891, p. 905. Nothnagel's spec. Path. u. Ther., Th. ii. p. 312.

MULTIPLE NEURITIS OF UNKNOWN ORIGIN.

A certain number of cases of multiple neuritis develop after exposure to cold. A few develop after unusual exertion, and quite a number occur without any ascertainable cause.

It is true that the more closely so-called idiopathic or spontaneous cases are scrutinized, in the light of our knowledge that a great variety of causes are competent to produce multiple neuritis, the more likely we are to discover some cause. Thus, many cases which have been reported as idiopathic can now be traced to tuberculous, carcinomatous, gouty, rheumatic, or diabetic conditions known to exist in the patients, but not formerly supposed to have any causal relation to neuritis. There are numerous cases of "idiopathic" neuritis which are preceded by severe gastro-enteritis. Is it not possible that an acute intoxication or ptomaine poisoning capable of causing the gastro-intestinal symptoms may produce a general neuritis? This question, which has occurred to several recent writers on the subject, seems to be answered affirmatively by such observations as are collected by Remak from recent literature.

It is possible that in some of these "idiopathic" cases there may be acute infection as the basis of the neuritis, inasmuch as some of the cases develop with an acute febrile attack, a chill, temperature rising to 102° or 104° F., and continuing for several days with the general manifestations of the febrile movement, a rapid pulse, nausea and vomiting, diarrhœa, occasionally jaundice, malaise, catarrhal symptoms in the various mucous membranes, and a general condition of acute sickness, such as is indicative of an infection. Remak[1] has laid great stress upon these spontaneous cases and has argued from the general constitutional disturbance that they are always infectious. He cites cases by Strümpell, Rosenheim, and Putnam, which were attended by a marked swelling of the spleen, in support of this hypothesis of infection. I am inclined to agree with Raymond (*Leçons*, 1897) that very many cases of paralysis hitherto ascribed to spinal-cord disease, but resulting in recovery, are actually cases of this nature, such as the cases recently described by Dana[2] as acute ataxia, and many cases of so-called acute poliomyelitis in adults.

Symptoms. The symptoms arising in these cases do not differ in any particular manner from the symptoms occurring in alcoholic neuritis, though Schultze is inclined to divide the cases into three categories, according to the prevalence of sensory, motor, or ataxic symptoms. The general description, therefore, of the symptoms of multiple neuritis already given is sufficient for these cases, and if the points that are mentioned under the head of diagnosis (page 110) are carefully considered it will be possible to separate these cases from other affections producing somewhat similar symptoms. The cerebral nerves do not escape in these cases, and, in fact, any of the symptoms which have already been considered under the heads of alcoholic multiple neuritis, arsenical multiple neuritis, and diphtheritic multiple neuritis may develop in the course of these cases. In a few cases optic neuritis has been observed, though, as already stated, this complication is rare. Sometimes painful swelling of the joints has been noticed at the beginning or in the course of the cases of idiopathic polyneuritis, and it has been a matter of discussion whether such joint affection was secondary to the neuritis or an independent rheumatic state.

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[1] Nothnagel's spec. Pathol. u. Therapie, Bd. xi. Th. ii.

[2] Jour. Nerv. and Ment. Dis., 1901, p. 105.

Korsakow[1] has affirmed that cerebral symptoms, such as have already been described as occurring in alcoholic neuritis, namely loss of memory, or delusions of memory, or disturbances of memory, or even a state approaching dementia, may develop in the course of these cases. I have never seen these psychical symptoms, however, in any but the alcoholic types of the disease.

SENILE POLYNEURITIS.

A form of multiple neuritis occurring in old age and not produced by any of the known causes of neuritis, and not attended by any of the acute symptoms, has been observed. This type of neuritis has been described chiefly by Oppenheim.[2] I have seen several cases of this affection. Patients are usually persons over the age of seventy years and are often the subjects of arterial sclerosis. The symptoms develop slowly. There is increasing weakness, with numbness in the lower extremities, and then in the upper extremities; but the patients do not suffer from sharp pain or from tenderness along the nerves. There is a progressive condition of paresis, which is much more marked in the hands and forearms and in the feet and legs than in the proximal portion of the extremities, and is rarely attended by any symptoms in the nerves of the head, eyes, or face. In some cases atrophy has attended the paresis and there has been a diminution in the contractility of the muscles to both currents. Knee-jerks are lost. There is rarely any ataxia. Very frequently the general weakness leads to a tremor, which, however, may be considered as one of old age. In one case that I observed, where the neuritis occurred in a person who had long suffered from chronic arthritis, the joints that had previously been stiff and deformed became quite relaxed, so that very abnormal positions could be given to the fingers and toes and to the wrists and ankles, without any perception of pain. In this case there was a complicating gangrene of one leg. It remained for a long time after the symptoms of the neuritis had subsided and the paralysis disappeared. The fixed deformity of the joints returned with the subsidence of the paralysis. In spite of their age, these patients do not always die of the disease, and may recover quite completely. Stein[1] has recently recorded a case in which the muscles of the eyes were paralyzed for a short time.

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[1] Zeitsch. f. Psych., vol. xlvi. p. 475, 1890. Arch. f. Psych., vol. xxi. p. 669.

[2] Berliner klin. Wochen., 1893, p. 589.

In the treatment of this form of multiple neuritis the importance of good food should be remembered, and the free use of stimulants may be required.

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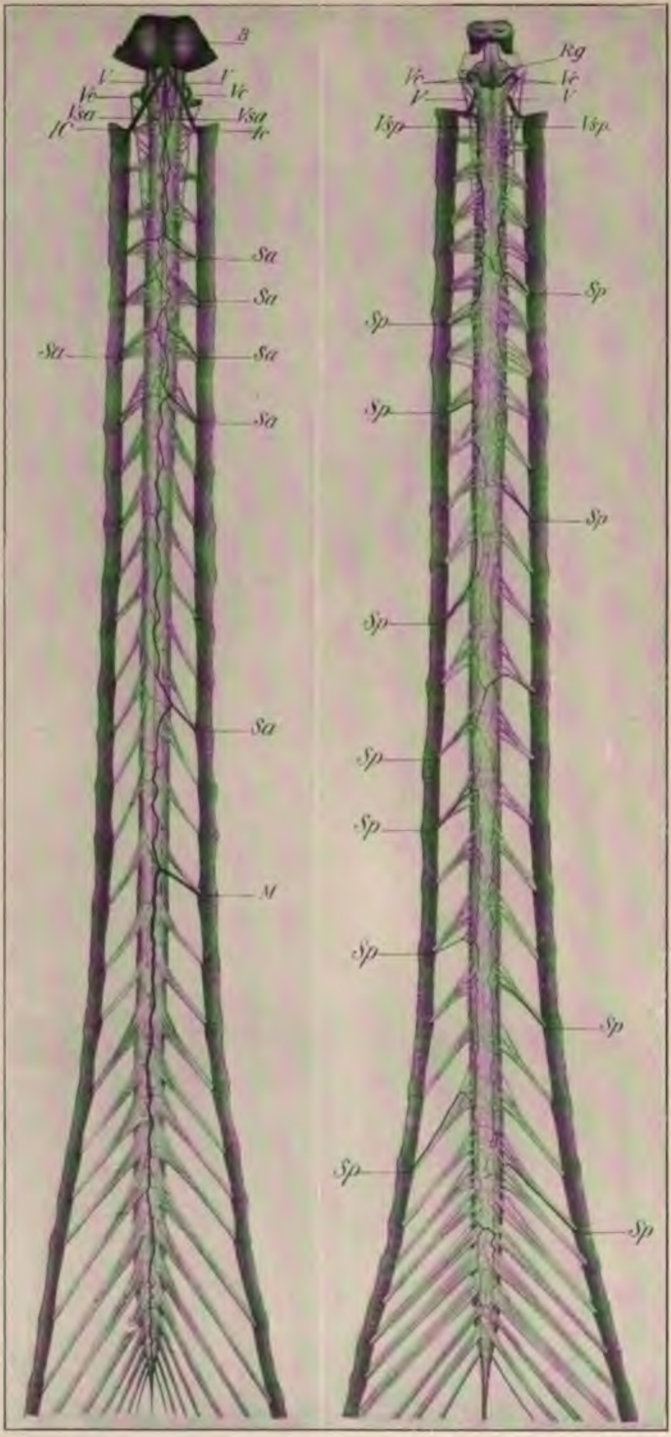
[1] Münchener med. Wochen., 1897, p. 463.



The Spinal Cord, its Nerves and Arteries. (Adamkiewicz.)

B. Basilar. *V.* Vertebral. *Vc.* Vertebro-cerebellar. *Vsa.* Vertebro-spinalis anterior. *Sa.* Spinalis anterior. *M.* Magna-spinalis. *Ic.* First cervical nerve. *Vsp.* Vertebro-spinalis posterior. *Sp.* Spinalis posterior. *Rg.* Fourth ventricle.

CHAPTER X.

DISEASES OF THE SPINAL CORD.

THE DIAGNOSIS AND LOCALIZATION OF SPINAL-CORD DISEASES.

THE spinal cord is a long cylindrical organ extending from the medulla oblongata at the base of the brain downward through the vertebræ to the level of the second lumbar spine. It is surrounded by two membranes—the pia mater, which contains its numerous bloodvessels, and the dura mater, which acts as a thick protecting sheath, and which lies against the bony walls without being closely adherent to them. From the sides of the spinal cord the spinal nerves come out in pairs, each nerve having two roots—an anterior root, which issues from the antero-lateral groove, and a posterior root, which enters the postero-lateral groove. On each posterior root lies an oval body—the posterior spinal ganglion—which contains the sensory neurone bodies. The cord has two enlargements, the cervical and lumbar, opposite to the exit of the nerves to the extremities. It is made up of thirty-one segments, each segment consisting of a mass of gray matter connected with a pair of spinal nerves which pass to a definite region of the body; and each segment is joined to the others and also to the brain by means of nerve tracts running through the various columns which surround the gray matter of the segment.

The spinal cord is an organ that has two distinct functions: namely, (1) the function of controlling directly the various parts of the body with which it is joined by means of its pairs of nerves; and (2) the function of conducting impulses to and from the brain. In considering, therefore, the functions of the cord and the symptoms that arise from disturbance of these functions, it is necessary to understand not only the function of each spinal segment so far as its motor mechanisms, its sensory connections, its vasomotor and trophic functions, and the distribution of its particular pair of spinal nerves, are concerned, but also the functions of the columns of the cord that pass through the particular segment concerned, and that transmit motor impulses downward from the brain and sensory impulses upward toward the brain.

In this chapter each symptom that may present itself in any case of spinal-cord disease will be considered, and these symptoms, one by one, will be referred to the anatomical structure whose function is impaired, and thus it will be possible to determine what the pathological import of each symptom may be. This will demonstrate how readily the localization of spinal affections may be determined, and

FIG. 32.

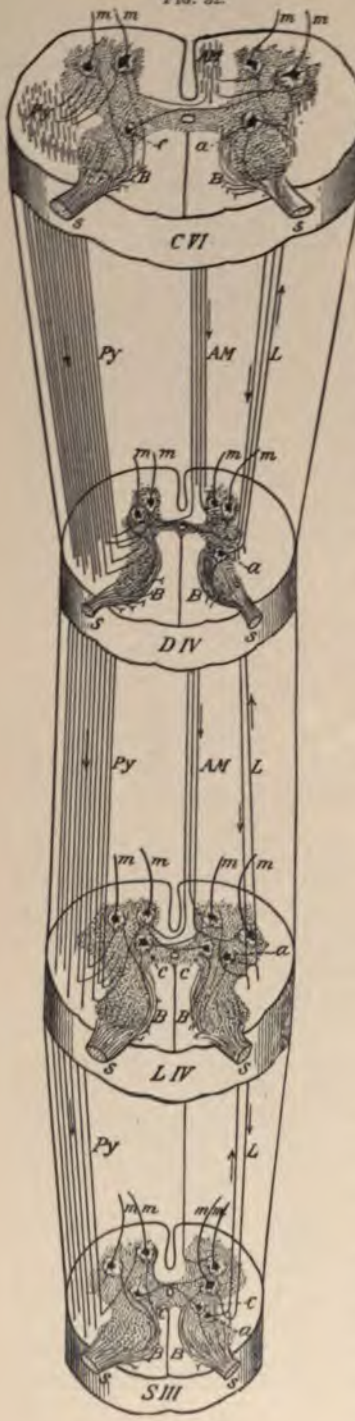


Diagram of the spinal cord, showing the motor mechanisms. *Py*, lateral pyramidal column; *AM*, anterior median column transmitting voluntary impulses from the right pyramid of the medulla to the motor cells of the anterior horns of the cord, whence motor nerves issue in the motor root (*m*); *S*, sensory nerve sending its fibres into the posterior horn and into the root zone of the column of Burdach (*B*), whence fibres pass forward to reach the commissural cells (*c*) and the association cells (*a*), and the motor cells (*m*). These form the motor mechanism of reflex and automatic acts. *L*, fibres of limiting layer, consisting of association neurones between various levels of the cord, being branches of the cells (*a*).

from this localization and the combination of the symptoms it will be easily possible to arrive at a diagnosis of any form of spinal-cord disease.

Symptoms. The symptoms of spinal-cord disease are paralysis, changes in reflex activity, disturbance in the control of the sphincters, alterations of gait and of posture, defects of sensation, ataxia, pain, and trophic disorders.

Paralysis is a condition of weakness or total loss of power in a muscle. It may be limited to a single muscle, it may affect a group of muscles, or it may affect a limb in its entire muscular apparatus. Paralysis may be due to an interference with the transmission of voluntary impulses from the motor centres of the brain to the motor cells (neurones) of the spinal cord, which impulses pass in the motor tracts of the spinal cord —viz., in the lateral pyramidal and anterior median columns. Paralysis may also be caused by a destruction of the motor neurones situated in the anterior gray horns of the spinal cord, whose axis-cylinder processes (axones) pass directly to the muscles through the anterior nerve roots and the motor nerves of the body. There are, therefore, two forms of spinal paralysis, quite clearly distinct from one another according as the lesion affects the first (cortico-spinal) or the second (spinomuscular) parts of the motor mechanism.

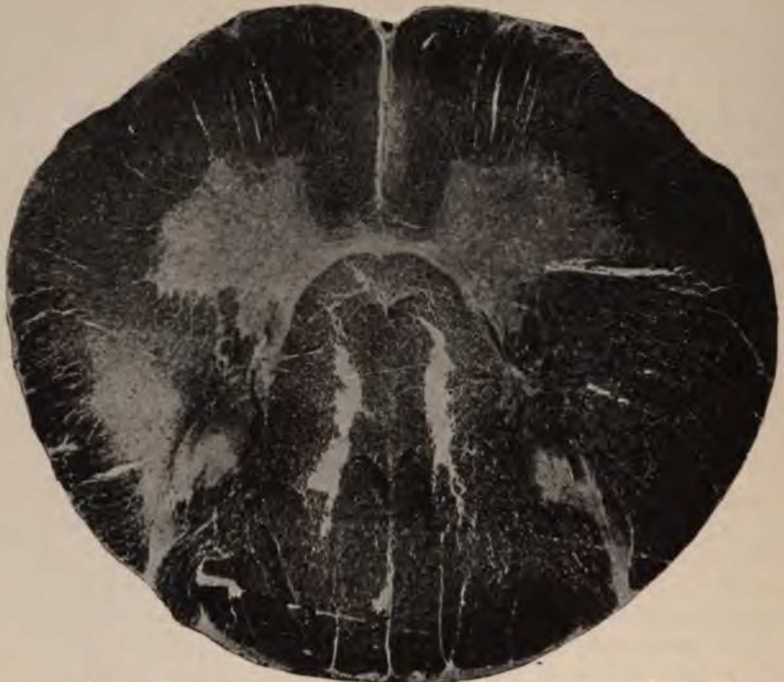
The diagram (Fig. 32) shows these two parts of the motor mechanism. The motor neurones of the brain send their axones downward through the pyramids of the medulla, where a partial decussation takes place, the majority of the axones from one pyramid crossing into the opposite lateral pyramidal tract of the cord (Fig. 32, *Py*), and the remainder passing directly into the anterior median oclumn. (Fig. 32, *AM*.) As these axones pass downward they terminate at various levels in the anterior gray horns of the spinal cord, their terminations taking the form of fine brush-like expansions which surround the motor neurones of the cord (see page 23), each filament coming into contact with the protoplasmic processes (dendrites) of those neurones, but not being continuous with those dendrites. As these motor axones pass downward to their termination they give off small fine branches (collaterals) at right angles to their course, and these collaterals terminate in the same manner in brushes around the motor neurones of the cord. Thus a voluntary impulse starting from a motor neurone of the brain may reach several motor neurones of the cord. As the motor tract passes downward through the cord it becomes smaller and smaller, but few fibres remaining in the anterior median column below the dorsal region, while some fibres of the lateral pyramidal tract extend to the very last segment of the cord.

This motor tract is much more highly developed in those animals which make use of the digits of their extremities. Thus in elephants the pyramidal tract is small, while in man it is most highly developed and large. Hence, it is evident that this tract transmits those voluntary impulses which are concerned in the finely adjusted movements of volition

Each axone in this tract, being a process of a motor neurone of the cerebral cortex, is dependent upon that neurone for its nutrition. If the cortical neurones are destroyed or if the axone is separated at any point in its course from its neurone, it degenerates to its terminal brush Hence a degeneration of the motor tracts of the cord may be due to disease in the brain, as in hemiplegia, or may be due to any transverse lesion of the cord which separates the axones from their neurones. The following figures (33 to 36) of specimens demonstrate descending degeneration of the motor tracts in the cord. Figs. 33 and 34 show descending degeneration consequent upon hemiplegia of slight and of severe type, the anterior median column on the side of the brain lesion and the lateral pyramidal tract upon the other side being degenerated. Figs. 35 and 36 show descending degeneration in both lateral pyramidal and anterior median columns consequent upon a transverse myelitis located at some distance above the level from which the section is taken.

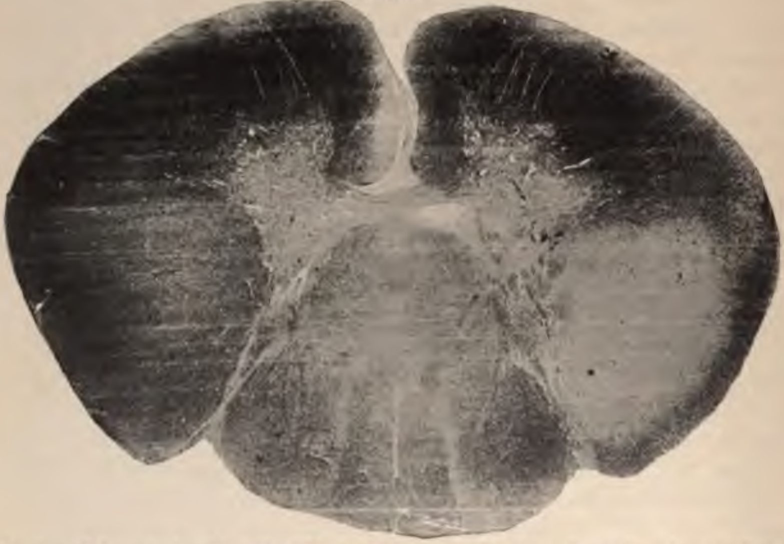
Paralysis of the cortico-spinal type, which is always due to a disease in this first element of the motor mechanism, has the following

FIG. 33.



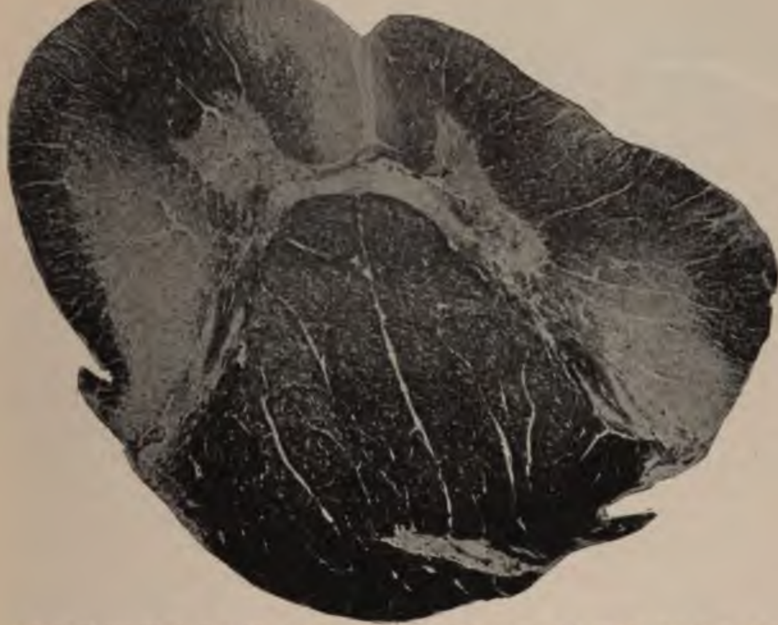
Section of the spinal cord, at the sixth cervical segment, showing descending degeneration in the left lateral pyramidal and the right anterior median columns, after a small lesion in the motor tract of the right cerebral hemisphere.

FIG. 34.



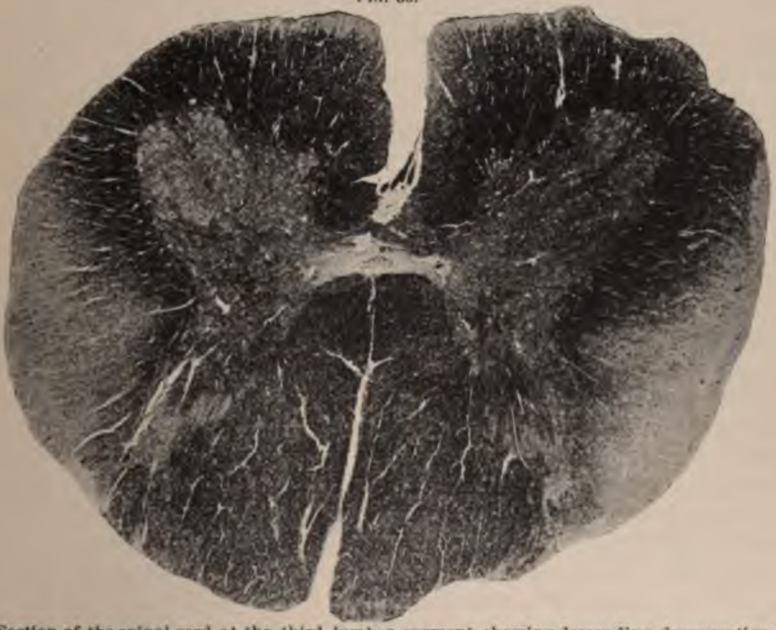
Section of the spinal cord, at the fifth cervical segment, showing descending degeneration in the left anterior median and right lateral pyramidal tract, after extensive lesions in the left cerebral hemisphere. There is slight degeneration in the left pyramidal tract and in the posterior columns.

FIG. 35.



Section of the spinal cord (somewhat distorted) in lower cervical region, showing bilateral descending degeneration in both anterior median and lateral pyramidal tracts. This section demonstrates the unequal size of corresponding columns on the two sides of the spinal cord. When the anterior median column is large in extent the opposite pyramidal tract is correspondingly small. Integrity of the direct cerebellar columns is evident.

FIG. 36.



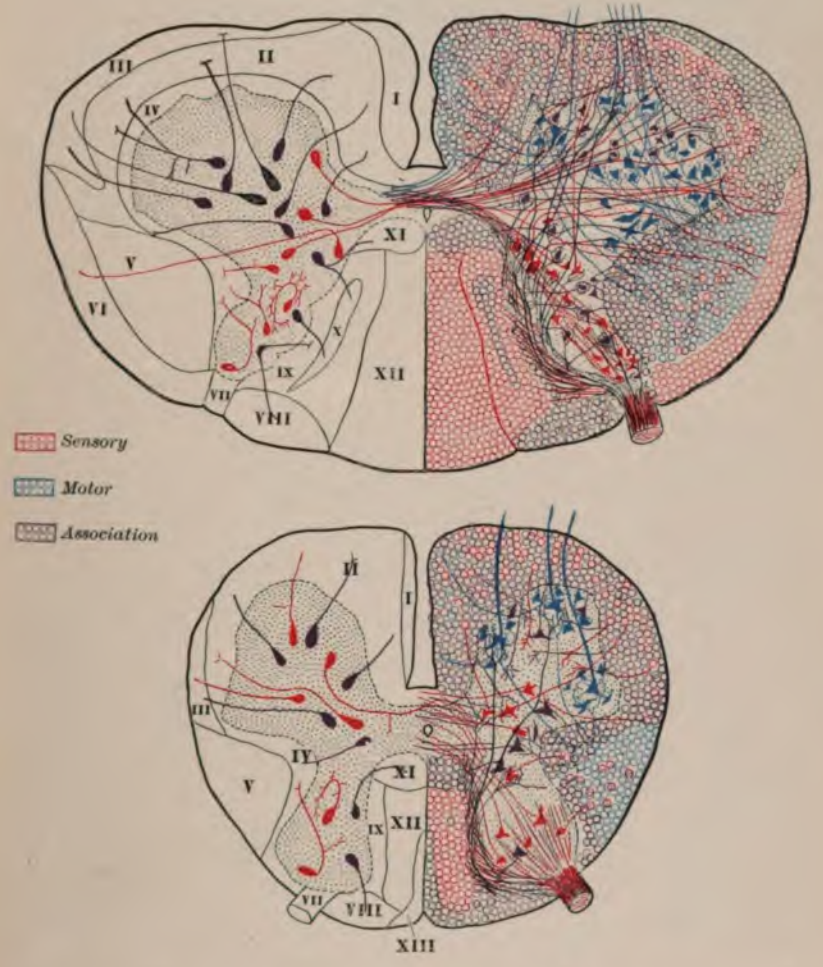
Section of the spinal cord at the third lumbar segment, showing descending degeneration in both lateral pyramidal tracts. A few fibres are degenerated in the anterior median columns. At his level there are no direct cerebellar columns.

characteristics: The muscles paralyzed are partially and not absolutely paralyzed. All the muscles of the limb affected are about equally involved; the limb is stiff, its joints being moved with difficulty and slowly on account of the rigid condition of the muscles. The muscles are thus in a state of slight tonic contraction and are hypersensitive to mechanical irritation, so that tapping a muscle produces a quick contraction, and tapping its tendon causes a prompt response. The muscles show no tendency to atrophy, though from disuse they may gradually become somewhat thin. They are never flabby; their nutrition is good; there is no change in their electric excitability. In this form of paralysis the circulation of the limb is sometimes impaired. There is slight blueness and coldness in the extremity and a tendency to œdema. Sensory disturbances do not necessarily attend this form of paralysis, and, if they are present, are an indication of disease elsewhere than in the motor tract.

This type of paralysis is seen after all forms of cerebral disease, and then usually affects the arm and leg upon one side (hemiplegia). It is also seen in both lower limbs after a transverse lesion of the cord, such as may be produced by Pott's disease, by transverse myelitis, by softening from thrombosis, by hemorrhage, or by tumors of the spinal cord. If this transverse lesion is in the cervical region, the arms are also involved. It is also seen in primary lateral sclerosis and in syphilitic paraplegia, in which conditions it is limited to the legs.

The second or spinomuscular type of paralysis has an entirely different set of characteristics. The muscles affected are usually entirely paralyzed at the outset, and if they recover at all, recover slowly and imperfectly. While all the muscles of the limb may be affected, as a rule they are not; a few muscles here and there upon the limb being greatly paralyzed, while others retain their power; or if all the muscles are paralyzed at first, some will recover to a greater extent than others. The limb is never stiff, but hangs helpless, yielding to the force of gravitation, its joints being relaxed and the articular surfaces no longer being held in close approximation by the tonic contraction of the muscles; hence these joints are all more freely movable than in health. The muscles are relaxed and flabby. They do not respond to mechanical irritation by tapping with a hammer, and tapping their tendons does not cause contraction; the so-called tendon reflex is lost. The muscles atrophy very soon after they are first affected, and this atrophy may go on rapidly until but little of the muscle is left. There is an early appearance of the reaction of degeneration in the muscle; that is, it no longer responds when either faradic or galvanic excitation is passed through its nerve, and it no longer responds when faradism is applied directly to it. The circulation in the limb is always impaired in this form of paralysis. The vessels are relaxed, the blood pressure is decreased, there is a slow capillary circulation, the limb is blue and cold, and all the chemical processes seem to be delayed; hence the surface tempera-

PLATE XII.



The Cervical and Sacral Enlargements of the Spinal Cord in Cross-section—showing the various neurones in the gray matter, the direction of their axones, and the varieties of fibres in the different columns of the cord. Blue, motor-; red, sensory-; purple, association- neurones and axones.

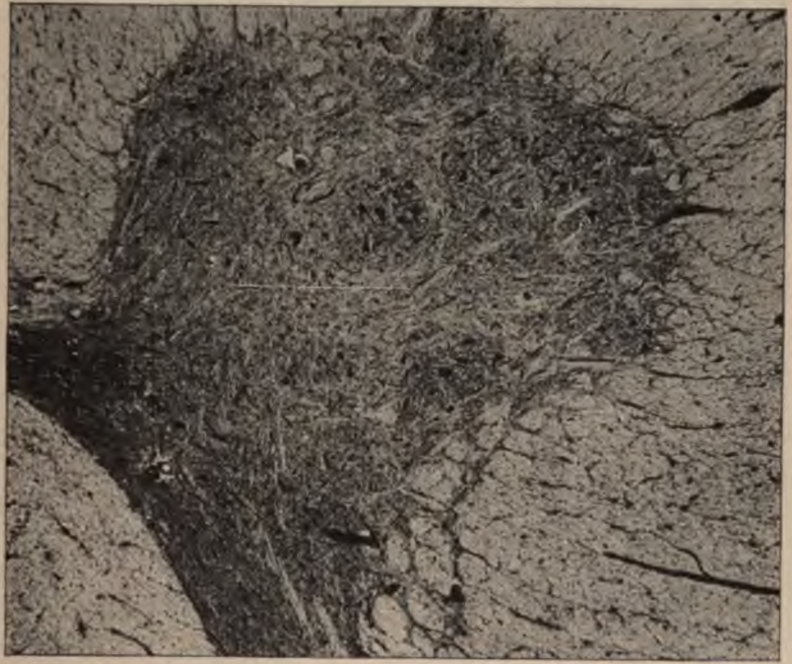
I. Ant. median column. II. Antero-lateral column. III. Gowers' antero-lateral ascending column. IV. Marginal column. V. Lateral pyramidal column. VI. Direct cerebellar column. VII. Lissauer's tract. VIII. Ext. portion of column of Burdach. IX. Root zone of the column of Burdach. X. Descending comma-shaped bundle of Schultze. XI. Post. commissural tract. XII. Column of Goll. XIII. Septo-marginal tract.

ture is much colder than in the first type of paralysis. Soon after the onset the limb is likely to be covered with clammy perspiration, and later on, when this is no longer present, it is very difficult to heat it up to the temperature of the other side. Sensory disturbances do not necessarily attend this form of paralysis.

This type of paralysis is due to a lesion of motor cells in the anterior gray horns of the cord (Fig. 32, *m*) or to an affection of the nerve trunks containing the axones of these motor neurones. It occurs in infantile spinal paralysis or anterior poliomyelitis, acute or chronic; in amyotrophic lateral sclerosis; in myelitis, either localized in a few segments or extensive throughout the cord; in syringomyelia when the lesion invades the anterior horns; in tumors and hemorrhages within the cord; and in softening of the cord due to embolism or thrombosis. This type is also present in neuritis.

The reason for these characteristics of this second type of paralysis will be better understood if the anatomical structure of the second part of the motor tract is considered. The motor neurones of the cord, as already stated, lie in the anterior part of the gray matter. These motor neurones are not scattered irregularly through its gray matter, but are collected into groups, the larger number of groups lying in the cervical and lumbar enlargements. The number of these

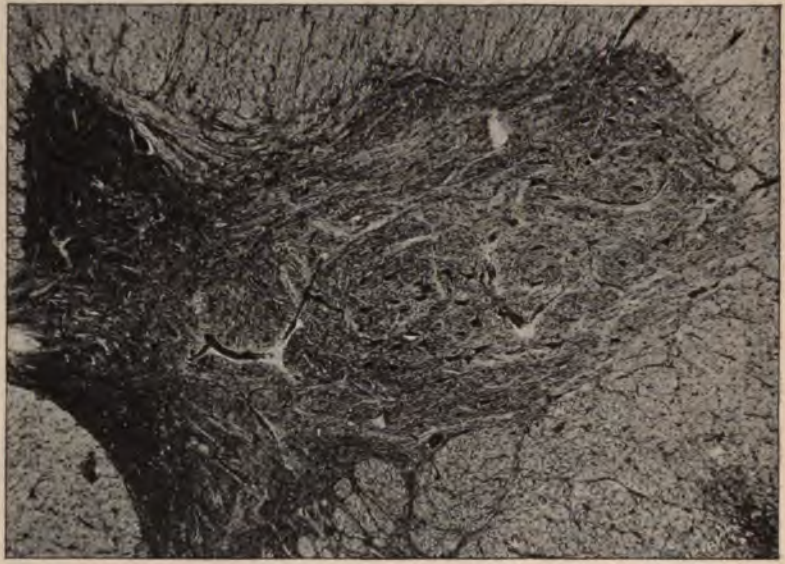
FIG. 37.



The groups of cells in the spinal cord at the fifth cervical segment.

groups varies greatly in different segments of the cord, as is shown in Figs. 37 to 39, and the groups have a varying extent 'ongitudinally, so that while some groups are limited to a single segment, others extend through several segments. Each group of neurones controls a single muscle or group of muscles which act simultaneously and in harmony. The fundamental movements of flexion and extension of the larger muscles of the limbs are represented in the large groups of the median, anterior, and lateral parts of the anterior horn. The accessory movements of the fingers and toes which are peculiar to monkeys and to man are represented in the smaller groups lying nearer the central portion of the gray matter. In Figs. 40 to 41 groups of cells at different levels of the cord are shown and it will be seen that they differ in their situation and extent at different levels. A careful study of comparative anatomy and of the lesions occurring in anterior poliomyelitis, in which disease single groups of cells are affected, has enabled us to determine the exact level in the cord of the various groups of cells representing the various muscles of the body. These are shown in Table I., which gives each segment of the cervical, lumbar, and sacral regions, with a list of the muscles represented in each segment. It will be noticed that some muscles are represented in two or even three segments, while other muscles are represented in but one. It is evident, therefore, that if the lesion in the cord is limited to one segment, it will paralyze completely two or three muscles which are represented by motor neurones in that

FIG. 38.



The groups of cells in the anterior horn of the spinal cord at the seventh cervical segment.

segment only, and it will paralyze partially other muscles which are represented not only in that segment, but also in adjacent segments. Hence the apparent irregular distribution and degree of the paralysis in various muscles upon the limb in cases of infantile paralysis.

TABLE I.—*Showing the Muscles Represented in Groups of Cells in the Various Segments of the Spinal Cord.*

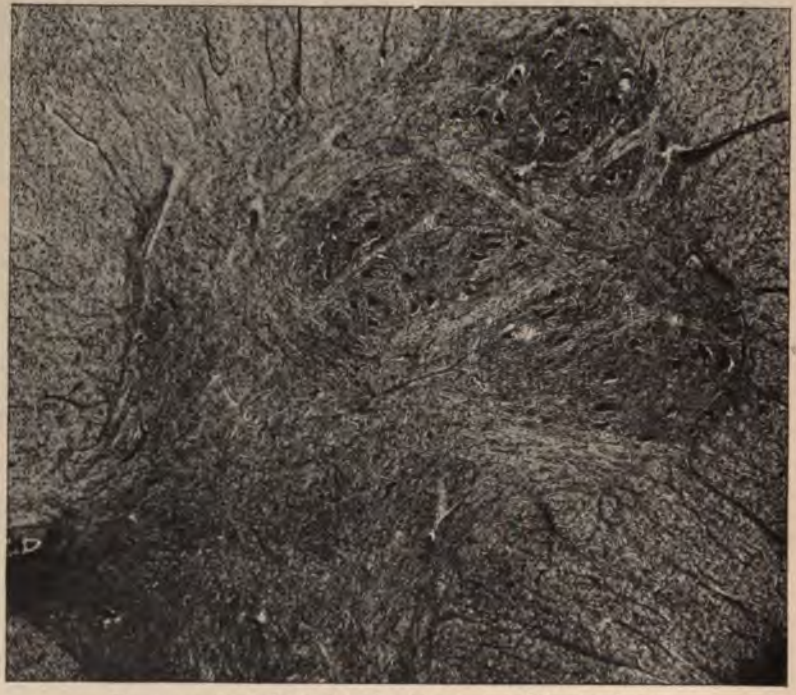
II., III. Cervical.	IV. Cervical.	V. Cervical.	VI. Cervical.	VII. Cervical.	VIII. Cervical.	I. Dorsal.
Diaphragm. Sterno-mastoid. Trapezius. Scalenus.	Diaphragm. Lev.ang.scap. Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps.	Rhomboid. Supra- and infraspin. Deltoid. Supin. long. Biceps. Supin. brev. Serratus mag Pect. (clav.). Teres minor.	Biceps. Serratus mag. Pect. (clav.). Pronators. Triceps. Brach. ant. Long extensors of wrist.	Pronators. Triceps. Brach. ant. Long extensors of wrist and fingers. Pect. (costal) Latis. dorsi. Teres major. Long flexors of wrist and fingers.	Long flexors of wrist and fingers. Extensor of thumb. Intrinsic muscles of hands.	Extensor of thumb. Intrinsic muscles of hands.

I. Lumbar.	II. Lumbar.	III. Lumbar.	IV. Lumbar.	V. Lumbar.
Quadr. lumb. Obliqui. Transversalis. Psoas. Iliacus.	Psoas. Iliacus. Sartorius. Quad. ext. cruris.	Quad. ext. cruris. Obturator. Adductores.	Obturator. Adductores. Glutei.	Glutei. Biceps femoris. Semi-tend. Popliteus.

I. Sacral.	II. Sacral.	III. Sacral.	IV. and V. Sacral.
Biceps femor. Semimemb. Ext. long. dig. Gastroc. Tibialis post.	Gastroc. Tibialis post. Tibialis anticus. Peronei. Intrinsic muscles of foot.	Peronei. Intrinsic muscles of foot.	Sphincter ani et vesicæ. Perineal muscles.

Certain special forms of the second type of paralysis are quite commonly recognized. Thus we have in anterior poliomyelitis the upper-arm type of paralysis, in which the deltoid, biceps, supinator longus, and muscles about the shoulder-blade are affected together, the muscles moving the wrist and fingers escaping. Reference to the table will show that this form is due to a lesion in the upper part of the cervical enlargement. We also have a lower-arm type of paralysis, in which the muscles which move the fingers and wrist are alone invaded, the supinator longus, which lies among them, escaping entirely. Reference to the table will show that this is due to a lesion of the lower cervical enlargement. The same distinction can be made in paralysis of the leg, there being a thigh type, a leg type, and a foot type, according as the lesion is in the upper lumbar, midlumbar, and sacral segments. By reference to the table, therefore, it will be possible in any case of paralysis of the second type to arrive at a localization of the lesion or at an estimate of its extent in the cord when the muscles paralyzed are exactly determined. The electrical examination will assist in this determination, inasmuch as the muscles whose groups of cells are destroyed will have lost their faradic excitability, while the muscles whose groups of cells are intact will show no change in electrical reactions.

FIG. 39.

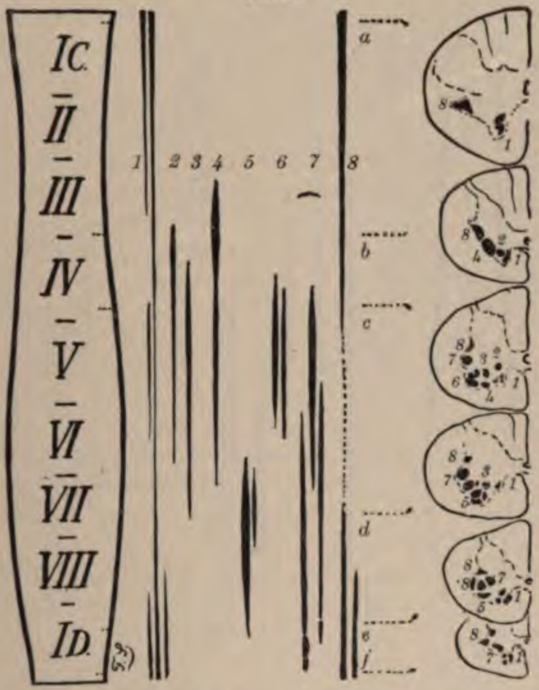


The groups of cells in the third lumbar segment of the spinal cord.

That the motor neurones of the cord have a distinct influence upon the nutrition of the muscle and upon the circulation in it, is shown by the rapid atrophy and the vasomotor disturbances in the muscle which also attend this type of paralysis, and to which allusion has already been made.

It has already been stated that this type of paralysis may be due to a lesion in the axones arising from the motor neurones of the cord.

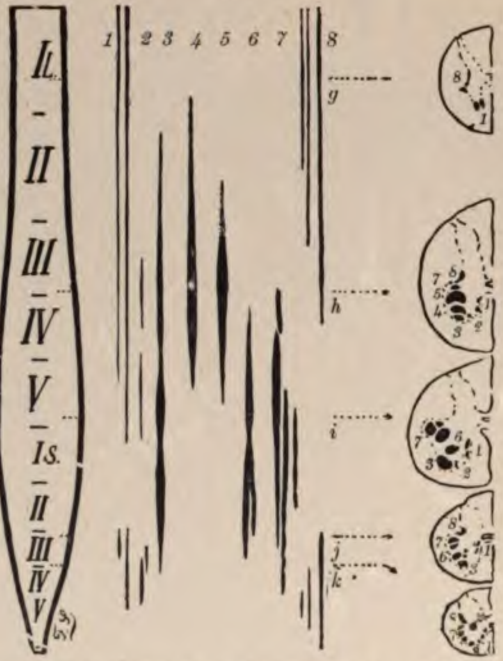
FIG. 40.



Columns of gray matter and motor nuclei of the cervical enlargement. Columna medialis—1. *a*, short rotators of head; M. subhyoid muscles; *b*, *c*, *d*, *e*, *f*, extensors and rotators of the vertebral column. 2, nucleus diaphragmatis (the series of sympathetic nuclei composed of small cells have not been drawn in); they are situated behind the columna medialis near the columna canalis centralis. Columna intermedio-lateralis—8. *a*, accessorius M. trapezius and M. sternocleidomastoideus; *b*, *c*, plexus cervicalis; *Mm*, trapezius sternocleidomastoideus; *d*, *e*, middle portion of M. trapezius; *f*, inferior part of M. trapezius; *e*, beginning of the nucleus for the M. latissimus dorsi. Columna extremitatis superioris—3. Mm. pectorales; 4. *b*, M. levator scapulæ; *c*, M. serratus major; 6, muscles of the shoulder; 7. *c*, M. biceps; lower down supinators and extensors of the fingers; between *d* and *e*, flexors and pronators; *e*, thenar and hypothenar muscles; *f*, hypothenar muscles; 5. *d*, M. triceps brachii; *e*, anconeus. (After F. Sano, Les localizations des fonctions motrices de la moelle épinière, Anvers, Belgique, 1898, p. 32.)

It is to be remembered, however, that after their exit from the cord these axones are divided up into nerves, being distributed in various directions through the brachial and lumbar and sacral plexuses. A differential diagnosis can always be made between lesions of the spinal cord and lesions of the nerve trunks by the distribution of the paralysis; for the muscles which are paralyzed together from a lesion of a single nerve are different from the muscles paralyzed together from a lesion of the spinal cord. Thus the deltoid is often paralyzed from a lesion of the circumflex nerve alone, but is never paralyzed alone from a lesion of the cord. Thus the extensors of the wrist, together with the supinator longus, are paralyzed in lesions of the musculo-spiral nerve; but, as already stated, these muscles are never paralyzed together in small lesions of the spinal cord, their groups of cells lying far apart. And what is true of the nerves of the brachial plexus is also true of the nerves going to the lower extremity. Hence, while the characteristics of paralysis in lesions of the spinal cord and of the motor nerve trunks are the same, the association of paralyzed muscles with one another in the two conditions differs. A further point of differentiation is also found in the distribution of anæsthesia occurring with lesions of the nerve trunks, anæsthesia being frequently present in lesions of the nerve trunks and not necessarily

FIG. 41.



Columns of gray matter and motor nuclei of the lumbar enlargement. Columna medialis—1. *g*, *h*, *i*. extensor and rotator muscles of the spine; *j*, *k*, musculus ischiococcygeus and M. levator ani; 2. *k*, in front M. sphincter vesicalis; behind *M*, M. sphincter ani; the sympathetic nuclei are not figured for the visceral muscles. Columna intermedio-lateralis—8. *g*, abdominal muscles; *h*, M. cremaster; *j*, *k*, muscles of the perineum. Columna extremitatis inferioris—2. *i*, M. pyramidales; 3. *h*, M. ilio-psoas; *i*, Mm. glutei; *j*, Mm, gemelli, M. pyriformis; 4. M. quadriceps femoris; 5. M. pectoneus; Mm. adductores; 6. *i*, flexors of the knee; lower down M. popliteus, M. triceps suræ, *j*; 7. *h*, M. tibialis anticus; *i*, extensor muscles of the toes, Mm. peronei; lower down M. tibialis posticus; flexors of the toes; *j*, *k*, intrinsic muscles of the foot. (After F. Sano, Les localizations des fonctions motrices de la moelle épinière, Anvers, Belgique, 1898, p. 33.)

present in lesions of the motor neurones of the cord. Furthermore, when it is present from a lesion of the cord, as will be shown later, the distribution of the anæsthesia in the skin differs entirely from the distribution of anæsthesia after a lesion of the nerve.

It is thus possible, from a study of paralysis and of its essential characteristics and distribution in any case, to reach a diagnosis and a localization of the lesion and to differentiate between lesions of the motor tracts of the cord or of the motor neurones of the cord or of the nerve trunks.

There are cases, however, of general disease of the cord, such as a general myelitis, involving both the white tracts and the gray matter, in which a third type of paralysis is observed. This presents some of the characteristics of both the former types, and, were the diagnosis to rest upon the examination of the paralysis alone it might present certain difficulties. Fortunately, there are other symptoms always present to guide us in the diagnosis; for in a general inflammation of the cord the changes in reflex action and in the control of the bladder and rectum, and the tendency to very severe trophic disturbances, are so marked as to leave no doubt regarding the actual condition. In this third type of spinal paralysis the paralysis resembles more closely the second type than it does the first, there being the same total paralysis, the same atrophy, and the same reaction of degeneration in the paralyzed muscles; but the distribution of the paralysis is more extensive—is, in fact, frequently total in both legs and almost complete in both arms, and, although there is no rigidity of the limbs, there is sometimes an increase in the reflex activity and in the mechanical excitability of the muscles in the early stage though later they are lost. This is particularly noticed in the disease amyotrophic lateral sclerosis in which both the lateral tracts and the anterior horns are simultaneously progressively destroyed. In this disease the first type of paralysis is present in the legs for several months, and the second type is present in the arms, associated with an increased mechanical excitability of the muscles, but finally this is lost, and the arms present the typical second type, which gradually and finally extends also to the legs.

In cases of injury of the spinal cord by fractures and dislocations of the vertebræ, the spinal cord may be seriously bruised or it may be absolutely disintegrated. The symptoms of paralysis appear to differ somewhat in these two conditions, as has been shown by Thorburn[1] and by Kocher.[2] When the spinal cord is injured, but not destroyed, there is total paralysis below the level of the injury, with a condition of rigidity of the limbs and an increase of tendon reflexes. There is also a loss of control of the bladder, which may take the form either of retention of urine or of spontaneous evacuation of the bladder. There is likely to be some distention of the abdomen by gas, due to a paralysis of the intestinal wall.

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[1] A Contribution to the Surgery of the Spinal Cord, Philadelphia, 1893.

[2] "Die Verletzungen der Wirbelsaule, zugleich als Beitrag zur Physiologie des Menschlichen Rückenmarkes," Mittheilungen aus den Grenzgebieten der Medizin und Chirurgie, Jena, 1896, vol. i. p. 401–460.

When the spinal cord is absolutely divided or destroyed at any level, there is total paralysis below this level, the limbs being completely relaxed and not in a state of rigidity. The paralysis is symmetrical upon the two sides. The tendon reflexes are absolutely lost. There is always a retention of urine, which has to be relieved by catheter; there is tympanites with distention of the abdomen; there is a paralysis of the vasoconstrictors leading to a dilatation of the subcutaneous veins, and consequently to an increased temperature, and to priapism. There is an increase in the genital reflex, obtained by pinching the testicle. There is a loss of sensibility to pain and temperature, and usually to touch also, at a definite level of the surface, as shown in Plate XIII. In any case in which these characteristics are absent after an injury of the spine, it is certain that the cord has not been completely destroyed at the level of the lesion.

Spasmodic contractions of the muscles of an involuntary kind are associated with paralysis in many forms of spinal-cord disease. Like the paralysis, they can be divided into two categories. When the condition present is spastic paralysis and the muscles are rigid and reflexes exaggerated, patients often notice a spontaneous trembling of the entire limb, due to alternate contraction of the two sets of opposing muscles. This is usually attended by an extensor spasm, so that the legs are stiffened and shake more or less violently. The condition is occasionally so extreme as to warrant the term "spinal epilepsy," first applied to it by Brown-Séquard, although this term is misleading and should be discarded. This symptom is seen in lateral sclerosis from any cause, and is indicative of a lesion in the lateral columns of the cord. When it is present the lesion does not involve the spinomuscular element of the motor tract.

Fibrillary twitchings of the muscles are much less painful than spasm of the entire muscle, but give considerable discomfort. Such fibrillary twitchings are present in diseases of the anterior horns of the cord, especially in chronic anterior poliomyelitis and in syringomyelia. Individual fibres of the muscle alternately contract, producing a little wave-like movement which goes on in the muscle itself. This does not involve a sufficient number of the muscle fibres to produce any contraction of the muscle as a whole. Such fibrillary twitchings can be elicited by percussion of the muscle or by exposure of the limb to cold. They always indicate a disease in the motor cells controlling the muscle, as they are absent in all forms of muscular dystrophy in which the disease is exclusively a muscular affection; hence such fibrillary twitchings are always associated with the second type of paralysis. They occasionally accompany the third type of paralysis, in which the motor cells are also affected.

Disturbance of reflex action is a symptom of great importance in spinal-cord diseases, the increase in reflex activity or the suspension of reflex activity being both of great significance. The diagram (Fig. 32) shows the anatomical basis of a simple reflex act. Whatever theory may be held with regard to the exact nature of the tendon reflexes, whether they are due to pure mechanical irritation of the fibres of the muscle, whose tone is maintained by spinal impulses, or whether they are due to a transmission of impulses through the spinal cord, the facts here stated hold true.

A reflex act is the immediate result of a sensory impression received in the spinal cord, and it takes place without necessarily producing any conscious perception and without any voluntary guidance. The sensory nerve enters the spinal cord through the posterior nerve root and divides into two parts, which separate in a Y-shaped division, one branch passing upward and the other downward in the root zone or column of Burdach (see Fig. 32, *B*). As these branches pass up and down they give off at right angles to their course small twigs (collaterals), and these collaterals, as well as the terminal filaments of the branches, terminate in brush-like expansions in the gray matter of the cord at various levels. Thus a sensory impulse entering in a posterior nerve is distributed to a considerable extent of the gray matter of the cord. Some of the terminal filaments pass forward to end about the motor neurones of the anterior horn of the same side upon which they enter; others pass forward and cross through the posterior commissure to terminate about the motor neurones in the anterior horn of the opposite side; these subserve reflex motor acts. Others terminate about the large cells in the median gray matter, which are the intrinsic or association cells of the cord, and transmit the impulses to other levels where they reach other motor cells (Fig. 32, *a*); others end about cells whose function is to control vasomotor and trophic reflex acts. Thus a single sensory impulse entering the cord may be widely distributed and awaken many reflex acts. Impulses entering the cord through the sensory nerve, and thus reaching various mechanisms of the cord, set up an activity in the various cells presiding over these mechanisms, and hence motor impulses pass outward to the muscles or to the viscera and result in their contraction, and hence in motion. The number of these reflex acts constantly going on in the body is enormous. In fact, in many of the lower animals the spinal cord is so much more developed than the brain, that it is evident that almost the entire nervous mechanism acts without conscious perception or voluntary control. The whole regulation of nutrition, of circulation, of digestion, of reproduction, and of excretion is regulated by the spinal cord independently of the brain. This is proven by the fact that in man, when consciousness is suspended in sleep, in coma, or by extensive injuries of the brain cortex, as in paresis and senile dementia, these vegetative functions go on in a normal manner. And it is well known that infants born with defective brains, or with almost no brain at all, may live for several months.

While the majority of these reflex acts are known to have mechanisms in the cord, there are only a few reflexes that are recognized and that can be tested in health and disease. These reflex acts are, first, the tendon reflexes; second, the skin reflexes; third, the automatic functions of the bladder and rectum.

(*a*) *Tendon reflexes* are produced by tapping the tendon of a muscle near its insertion and thus producing a sudden contraction of the muscle. Thus the tendons about the wrist and elbow, the patella tendon at the knee, and the Achilles tendon at the ankle can be excited in a state of health. We now know the exact level in the spinal cord that contains the reflex mechanism necessary to the existence of these various reflex acts. In Table II. the various spinal muscular reflexes are given, the method of producing the reflex, and the level of the segment controlling each.

TABLE II.—*Localization of Muscular Reflex Acts in the Spinal Cord.*

Reflex acts.	Localization in segment.
Pupillary reflex through the sympathetic: Dilatation of the pupil produced by irritation of the neck.	Fourth cervical to first dorsal.
Scapular reflex: Irritation of the skin over the scapula produces contraction of the scapular muscles.	Fifth cervical to first dorsal.
Biceps and supinator longus: Tapping their tendons produces flexion of the forearm.	Fifth and sixth cervical.
Triceps reflex: Tapping tendon produces extension of forearm.	Sixth cervical.
Scapulohumeral reflex: Tapping the inner lower edge of the scapula causes adduction of the arm.	Seventh cervical.
Tapping extensor tendons at the wrist causes extension of the hand.	Sixth to eighth cervical.
Tapping flexor tendons at the wrist causes flexion of the hand.	Seventh to eighth cervical.
Palmar reflex: Stroking palm causes closure of fingers; finger clonus.	Eighth cervical to first dorsal.
Abdominal reflex: Stroking side of abdomen causes retraction.	Ninth to twelfth dorsal.
Genital reflex: Squeezing the testicle causes contraction of the abdominal muscles.	First to third lumbar.
Patella tendon: Striking tendon at knee causes extension of the leg; "knee-jerk."	Second and third lumbar.
Foot clonus: Extension of Achilles tendon causes flexion of the ankle.	First to third sacral.
Plantar reflex: Tickling sole of foot causes flexion of toes, or extension of the great toe and flexion of the others.	First to third sacral.

In the process of disease these reflex acts may be lost or they may be exaggerated.

Loss of Tendon Reflexes. A loss of the reflex implies a lesion in the reflex mechanism, either in the sensory nerve which would necessarily result in a coincident condition of anæsthesia, or in the sensory reflex fibres within the cord, which might give rise to this symptom alone, or in a destruction of the motor mechanism, which would give rise to a coincident condition of paralysis. If, therefore, by the distribution of the anæsthesia or of the paralysis it is possible to exclude in any case a lesion of the nerve trunk, the loss of reflex necessarily implies a lesion of the spinal cord. This lesion will necessarily be limited to the segment controlling a reflex tested. Therefore, in the examination of any case of spinal disease, it is incumbent to try each of the reflexes in turn, and, if any one of them is lost, to direct particular care to the examination of the other functions of the segment of the cord in which the lesion is indicated. The diseases in which the reflex activities are suspended are locomotor ataxia from the very earliest stage, and also those cases of general paresis in which there is an early complicating sclerosis of the posterior columns of the cord; syringomyelia, anterior poliomyelitis, general myelitis in the later stages, transverse myelitis at the level of the lesion, disseminated sclerosis when the patch of sclerotic tissue happens to lie in the reflex arc, tumors and hemorrhage in the cord at the site of the lesion. It is evident that a loss of tendon reflexes is a common accompaniment of the second type of paralysis.

An exaggeration of the tendon reflexes is also frequently observed as a symptom of spinal disease. This implies a suspension of the inhibitory impulses coming from the brain in a state of health, which impulses pass downward through the lateral columns in the motor tracts. The inhibition being removed, the spinal cord reacts more quickly and intensely to sensory impulses coming into it from without. In this condition a phenomenon known as clonus may be elicited in almost any one of the tendons of the longer muscles. Clonus consists of a rapidly repeated series of contractions in a muscle set up by a sudden overextension of the tendon of that muscle. Thus if the patient's leg be allowed to rest upon the left hand of the examiner, and the right hand grasping the foot presses it suddenly backward, thus extending the Achilles tendon, if ankle clonus is present a series of vibrations of the foot will be produced, due to a repeated contraction of the muscles of the calf of the leg. A similar clonus may be elicited in the quadriceps femoris by a forcible pressure downward upon the patella tendon when the limb is relaxed and extended. A similar clonus may occasionally be elicited in the tendons of the elbow, of the wrist, and of the fingers.

A symptom known as Babinski's reflex is always to be elicited when the tendon reflexes are exaggerated, and is a positive sign of some disease in the lateral column of the cord or in the motor tract. This is a sudden extension of the great toe, produced by scratching the sole of the foot, due to a contraction of the extensor muscle, which stands out like a cord. It does not occur in hysteria, in which other reflexes may be increased; hence it is a valuable diagnostic symptom.

The existence of clonus, like that of exaggerated reflexes, is an indication of disturbance of function in the lateral columns of the spinal cord, and is, therefore, commonly associated with the first type of spinal paralysis. It is also an early indication of any pathological process which interferes with the transmission of motor impulses from the brain to the cord. Thus in the early stage of Pott's disease, when slight pressure is made upon the cord or some disturbance of the nutrition of the cord occurs opposite the caries, the reflexes are commonly exaggerated in the segments below. Exaggeration of the reflexes is present in lateral sclerosis, in descending degeneration of the lateral columns below the level of the lesion, in syphilitic paraplegia, and in the early stage of acute myelitis when the pathological process irritates the motor neurones before they are destroyed. It is particularly noticeable in the arms in amyotrophic lateral sclerosis in the early stage of the disease, but as the process advances reflexes are lost in the arms, while they continue to be exaggerated in the legs. An increase of tendon reflexes is not infrequently observed in hysteria.

While it is true that a compression of the spinal cord of slight or intense degree, or a partial destruction of the spinal cord in its upper region, always produces an increase of the tendon reflexes in the parts below the lesion, it has been observed by Miles and Bastian that in some cases, where, by injury (fracture or dislocation of the vertebræ), the spinal cord has been entirely divided or so completely crushed that all its elements have been destroyed, the tendon reflexes below the point of destruction have been abolished. This is not thought to be due to the concussion of the cord consequent upon the injury, as it may persist for many weeks. It has been ascribed to a setting up of abnormal inhibitory impulses from the point of destruction downward, due to the irritation of the lesion. This, however, is not accepted by Bastian, and no satisfactory explanation of the condition can be offered. In any case of localized crush of the cord from an injury the absolute loss of tendon reflexes is thought to indicate a total destruction of the cord, and in such a case any operation at the site of injury would be futile.

(*b*) *The skin reflexes* are not as well understood as the tendon reflexes. When certain areas of the skin are slightly stroked or tickled, there occurs a slight movement of the skin at a part not under the point of irritation, but near to it, which seems to be due to the contraction of involuntary muscular fibres lying under the skin. These skin reflexes are best seen in animals, especially horses and cows, for the sting of a fly is capable of setting up a little fibrillary twitching in the skin of their bodies at almost any part. The following table (III.) gives a list of the skin reflexes found in man, the method of their production, and the level of the spinal cord that is supposed to control them, though it is not certain that they are under the control of a direct spinal mechanism:

TABLE III.—*Localization of Skin Reflexes in the Spinal Cord.*

Reflex acts.	Localization in segment.
Epigastric reflex: Stroking breast causes dimpling of the epigastrium.	Seventh to ninth dorsal.
Cremasteric reflex: Stroking inner side of thigh causes retraction of scrotum.	First and second lumbar.
Gluteal reflex: Stroking buttock causes dimpling in the fold.	Fourth to fifth lumbar.

The skin reflexes are usually lost in those diseases in which the tendon reflexes are exaggerated. They are also lost in brain diseases, but never in hysteria. They are never exaggerated.

The reflex mechanisms controlling the bladder and rectum are complex and are located in the fourth and fifth sacral segments of the spinal cord.[1] The sensory impulses setting up this mechanism come from the mucous membrane of the bladder or of the rectum, and, passing inward to the cord, produce two separate effects: one is the active motor impulse of contraction in the muscles which empty these organs; the other is an inhibitory impulse arresting the action of those muscles which normally exert a constriction at the opening of these organs. Such evacuation of their contents by a reflex act may occur without the knowledge of the individual or without his control when disease cuts off the lower part of the cord from its communication with the brain, as in a transverse myelitis of the dorsal region. We then have a condition known as active incontinence, in which the organs are emptied spontaneously at intervals in a normal manner. The mechanism itself, however, may be destroyed by any lesion of the sacral region of the spinal cord. Under these circumstances, the reflex arc being broken and the motor cells controlling muscular action being destroyed, the organs are no longer evacuated by reflex impulses, and their evacuation has to be attained by outside aid. In some individuals a distention of the bladder finally overcomes a constrictive action of the sphincters, and then there is a constant leakage, producing a passive incontinence of urine, but the rectum shows no tendency to empty itself. In some individuals the constrictive action of the sphincters seems to be unusually strong, and occasionally a distention will lead to a rupture of the bladder rather than to its evacuation by water pressure. Sometimes there is a permanent weakness of the sphincter and a constant dribbling of urine without any distention of the bladder.

Disturbances in the action of the bladder and rectum constitute the chief sources of danger in spinal-cord disease, for a retention of urine is liable to set up catarrhal conditions, and, finally cystitis, and the evacuation by means of a catheter is liable to lead to the infection of the bladder by germs unless the catheter is absolutely aseptic. Such a cystitis when once set up very often leads to pyelitis, nephritis, and death; hence the greatest care is to be taken of the bladder in spinal-cord disease. The evacuation of the rectum must also be carefully attended to, even though this involves its emptying by digital manipulation, as is commonly the case. Disturbance in the action of the bladder and rectum occurs in almost all forms of spinal-cord disease, and, therefore, is not diagnostic of any one special pathological lesion or of its location. It rarely occurs in hysteria, though retention is occasionally present.

The Gait in Spinal Disease. In various forms of spinal-cord diseases the position and gait assumed involuntarily by the patient in walking are abnormal. In diseases which produce the first type of paralysis—*e. g.* lateral sclerosis—the gait is a stiff one; the hips and knees are held rigid and adducted, so that in walking the knees appear to touch or even to overlap, and the feet are not lifted from the floor, and hence are dragged or are pulled forward with effort, the great toe scraping the floor. At the same time the tendency to ankle clonus frequently leads to trepidation of the entire body from the shaking of the foot. These patients cannot step out freely, and hence their steps are very short, but quite regular in length. They rise from a chair or sit down slowly, there being an apparent resistance to the flexion and extension of the joints. Their motions are not awkward and can be guided accurately. This has been named the spastic gait.

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[1] Van Gehuchten. Le Nevraxe, 1902, vol. iv. p. 119.

In diseases involving the lumbar enlargement of the cord and affecting the gray matter alone or the entire cord, and producing the second type of paralysis, the gait is the paralytic gait. As the muscles grow weaker the support of the joints becomes imperfect, so that the aid of canes and crutches is needed to support the hip, and there is a tendency for the knee to be overextended and for the ankle to turn. The paralysis of the muscles of the thigh makes the ordinary lifting of the feet from the floor impossible, and hence the leg is dragged along the floor, the inner side of the foot touching the floor, and the limb dragging rather loosely as it is pulled forward between the crutches. There is none of the stiffness of the spastic gait and none of the uncertainty of the ataxic gait. If the anterior tibial group of muscles is paralyzed completely, there will be a drop-foot, and if this precedes paralysis of the thigh or is alone present, then the patient in stepping forward lifts the leg higher than normally in order to avoid stubbing the toe. This has been termed the stepping gait, as the patient has the appearance of stepping over an obstacle in his way or of raising his legs as if about to go up stairs. Such patients are soon unable to rise from a chair or to ascend steps.

In posterior sclerosis (locomotor ataxia) the gait is uncertain, the steps being of irregular length, the patient not appearing to place the foot upon the ground in the position that he desires, and hence he loses his balance frequently and makes irregular efforts to preserve it. He rises with difficulty from a chair, spreads his feet far apart, and sways for a moment after rising, in order to get his balance. As the disease advances the gait becomes more irregular, with a tendency to long steps, to too great lifting of the foot from the floor, too sudden and violent placing of the foot downward upon the floor, so that the gait has been called a stamping gait. The feet are uniformly placed too far apart, as the patient seeks a wide base of support. The irregularity of the gait is increased by an attempt to walk backward or to walk forward with the eyes closed. This has been named the ataxic gait.

The Position Assumed in Bed. When the patient is confined to his bed after an injury of the spinal cord, such as occurs from fractures and dislocations, the position assumed in bed differs according to the level of the injury.

If the sacral segments are crushed, the patient is unable to move the feet and ankles, and hence the feet lie in an extended position, assuming the position of drop-foot. The thighs and knees can be moved in this condition.

If the lesion involves the lower half of the lumbar enlargement, the patient lies with the thighs drawn up, the legs flexed, and he is unable to straighten the legs voluntarily or to lift the feet.

If the lesion involves the entire lumbar enlargement, the patient lies with the thighs, legs, and feet extended, and cannot move them.

If the lesion is in the dorsal region, the lower extremities are paralyzed more or less completely, but there is no tendency to drop-foot, and the limbs offer some resistance to passive motion, the tendon reflexes being exaggerated and the muscles somewhat rigid, unless there has been a total destruction of the cord, under which circumstances the muscles are relaxed and the tendon reflexes lost.

If the lesion involves the first dorsal and last cervical segments the hands will be in a position of *main en griffe,* but the elbows and shoulders can be freely moved.

If the seventh cervical segment is destroyed, the forearms are partially flexed and lie upon the body with the hands pronated. Voluntary movements of the wrist are impossible, but the elbow and shoulder can be moved.

If the lesion lies at the sixth cervical segment, the arms are abducted from the side, the forearms are supinated, wrists and fingers being paralyzed.

If the lesion is at the fifth segment, the arms lie extended and relaxed at the side of the body, all motion being impossible.

These forced positions are assumed because of the fact that at any level of the cord the muscles controlled at and below the lesion will be paralyzed, and those controlled by the cells just above the lesion will be actively contracted by the state of irritation into which their motor centres are thrown, or if two centres of opposing action are irritated together, the stronger will overcome the weaker, and the resulting position, once assumed, cannot be corrected voluntarily; hence the fixed position once assumed is maintained.

Disturbances of sensation are important symptoms of spinal-cord disease. Any irritability of the sensory areas of the cord may lead to a hypersensitive condition of the skin, to the perception of ordinary impulses as extraordinary ones, and hence to the too keen appreciation of any sensation which may be started up. The irritation in the cord may be so great as to lead to hallucinations of sensation; that is, to the perception of sensations in the skin which are set up in the cord and do not really come from the skin (similar in origin to the tingling felt in the little finger on compressing the ulnar nerve at the elbow). Hence in any disease of the cord during the early stage of congestion, or in the irritation produced by beginning scle-

Fig. 42.

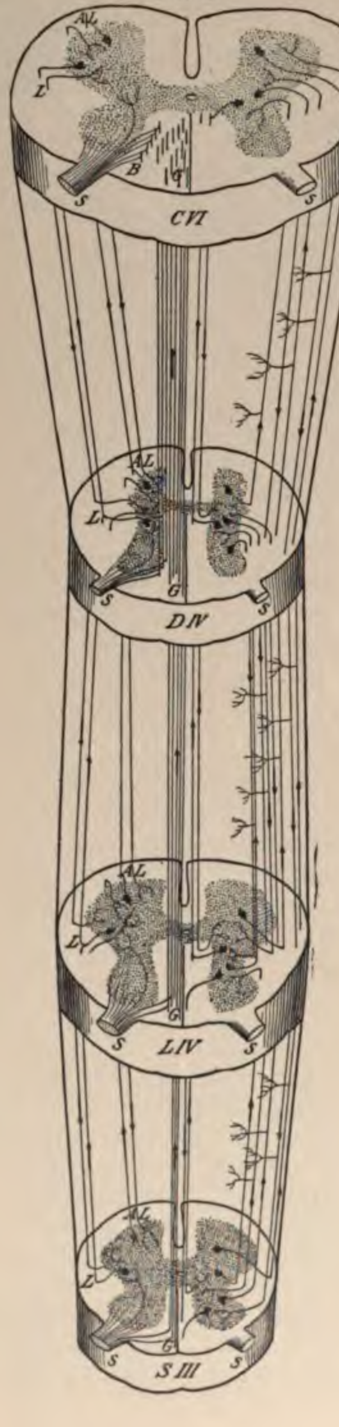
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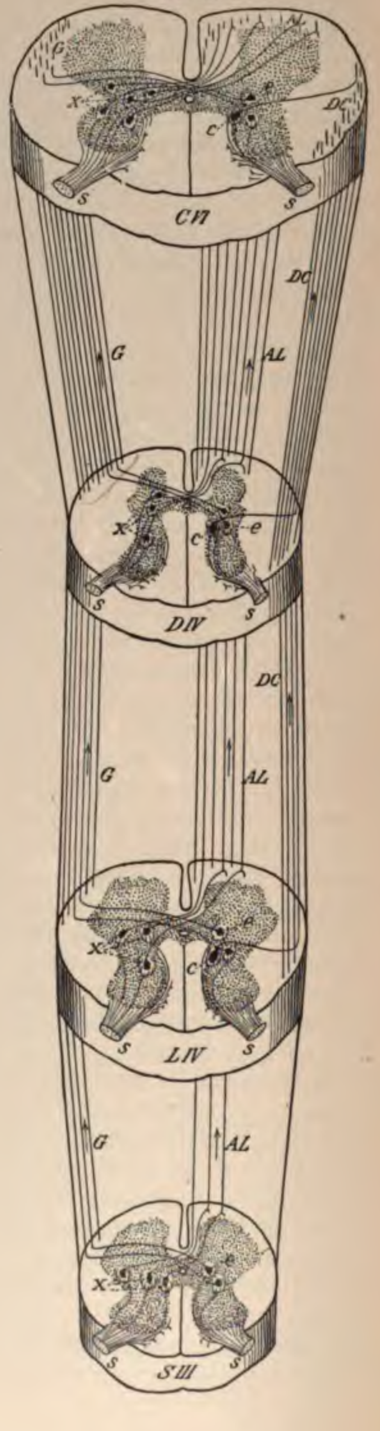
Fig. 43.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rosis, patients commonly complain of tingling and numbness, of sensations of burning or of cold, of sensations of pain, of fulness, of pressure, and of weight. These sensations are referred to the skin or to the limbs, and to particular parts of the skin and limbs that correspond to the segment of the cord which is irritated. These sensations are classed together under the term "paræsthesiæ."

Any destruction of the sensory areas of the cord will lead to a suspension of sensations which are classed together as anæsthesiæ. Sensations are of several kinds—namely, sensations of touch, the loss of which is tactile anæsthesia; sensations of pain, the loss of which is analgesia; sensations of temperature, the loss of which is thermo-anæsthesia; and sensations of muscular sense, the loss of which leads to imperfect co-ordination or ataxia. As these sensations reach the spinal cord through the sensory nerve roots, any disease of these nerve roots affects uniformly all forms of sensibility. But the sensory nerve roots transmitting these sensations separate one from another after their entrance into the cord, and pursue different courses in their way upward to the brain. Hence it is possible for one form of sensation to be lost while the others are preserved in diseases affecting different tracts in the spinal cord. Thus in diseases of the root zone of the column of Burdach, through which all sensations pass, we have anæsthesia, analgesia, and ataxia. In diseases of the central gray matter of the cord we have analgesia and thermo-anæsthesia without anæsthesia or ataxia. In diseases of the posterior columns of the cord we may have ataxia alone or associated with anæsthesia. The course of these various impulses into the cord and upward through the cord is not as yet absolutely determined, but so far as it is known it is shown in the diagrams (Figs. 42, 43).

The sensory fibres are the axones of neurones situated in the posterior spinal ganglia, and if they are cut off from connections with these ganglia by any lesion of the nerve root or any lesion within the cord, they will degenerate from the point of lesion onward. A

EXPLANATION OF FIGS. 42 AND 43.

FIG. 42.—Diagram showing long sensory fibres in the posterior columns of the cord: *S*, sensory nerves whose fibres turn upward after entering the root zone. Each successive nerve root from below upward presses the fibres already ascending inward and backward, so that in the cervical region the fibres which have come from the sacral region lie in the column of Goll near to the posterior septum; the fibres from the lumbar region lie in the column of Goll external and anterior to those from the sacral region; the fibres from the dorsal region lie at the lateral part of the column of Goll; and the fibres from the cervical region lie in the column of Burdach. This diagram also shows association neurones of the cord whose axones are passing upward and downward in the marginal portion of the posterior column and in the lateral (*L*) and antero-lateral (*AL*) columns of the cord, with thin collaterals.

FIG. 43.—Diagram showing the course of long sensory columns in the spinal cord: *S*, sensory nerve roots whose fibres enter the root zone and the gray matter. On the right side of the diagram these fibres terminate about the cells of the column of Clarke (*C*), whence fibres pass into the right direct cerebellar column (*DC*), and thus upward to the cerebellum. Sensory fibres also terminate about the intrinsic cells of the cord (*c*), whence fibres cross to the opposite side and ascend in the column of Gowers (*G*). On the left side of the diagram sensory fibres are seen to terminate around intrinsic cells of the gray matter (*X*), whence fibres cross over to the opposite side of the cord and ascend in the antero-lateral column (*AL*).

study of the degenerations following lesions of the nerve roots and following transverse lesions of the spinal cord has given us some knowledge of the course of the sensory tracts.

The fibres of the posterior nerve root on entering the cord divide in a Y-shaped manner, one branch turning downward and the other upward. The branch which turns downward is short. It descends in the portion of the column of Burdach lying anterior and external to the root zone, named the comma-shaped column of Schultze, and terminates by sending its fibres (collaterals) into the gray matter of the posterior horn. The special function of these fibres is unknown. The branches that turn up are of various lengths, and some fibres ascend all the way to the medulla; these are the long sensory tracts. The great majority of the branches which turn upward terminate soon after their entrance into the cord in the segment into which they enter or in the segments just above it; these are the short sensory nerve fibres. They terminate in branches around the cells of the gray matter in the posterior and anterior horns and in the central gray. A few of the fibres also turn upward in a small zone lying near to their entrance and between the tip of the posterior horn and the periphery of the cord known as Lissauer's column.

The course of the long sensory tracts is pretty well known, and is well illustrated in the figures here given (Figs. 42 and 43). When a single nerve root is injured or destroyed the area of degeneration at its point of entrance into the cord is quite extensive, but at higher levels in the cord the area of degeneration grows smaller, and at the junction of the cord with the medulla it is quite limited in extent. This is well illustrated in Figs. 44 to 46, which show the area of degeneration in a case of unilateral tumor involving the second and third lumbar nerve roots.[1] It will be seen that at the second lumbar segment the entire nerve-root zone in the column of Burdach is degenerated. In the mid-dorsal region this degeneration is limited to a small strand in the column of Goll, and in the cervical region to a small strand also in the column of Goll and near to the median line. This case confirms the results of physiological experiments in which the posterior roots have been divided in monkeys.[2] Such a case demonstrates that of the large number of nerve fibres entering in any one posterior nerve root but a few extend all the way up to the medulla. If a series of cases of transverse lesion of the cord is studied, the transverse lesion in different cases being situated at different levels, it is found that the ascending degeneration in these long fibres within the posterior columns of the cord varies in extent in different cases. The higher the transverse lesion the larger the area of degeneration produced. This is demonstrated in Figs. 47 and 48, showing the area of ascending degeneration following a lesion

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[1] Case reported by Osler. Journ. Nerv. and Ment. Disease, 1889, p. 499.

[2] Beiträge zur Anatomie des Centralnerven-systems insbesondere des Rückenmarkes, von Prof. Dr. J. Singer und Dr. E. Munzer in Prag, Wien, 1890.

of the lower lumbar region (Fig. 47), as contrasted with the area of degeneration following one in the upper dorsal region (Fig. 48). A study of such cases has made it possible to determine the relative position in the columns of Goll and Burdach occupied by the long sensory fibres coming up from the various segments below. This is shown in Fig. 42. While the lesion in the posterior root zone causes an ascending degeneration in the posterior columns only, it has been found

FIG. 44.

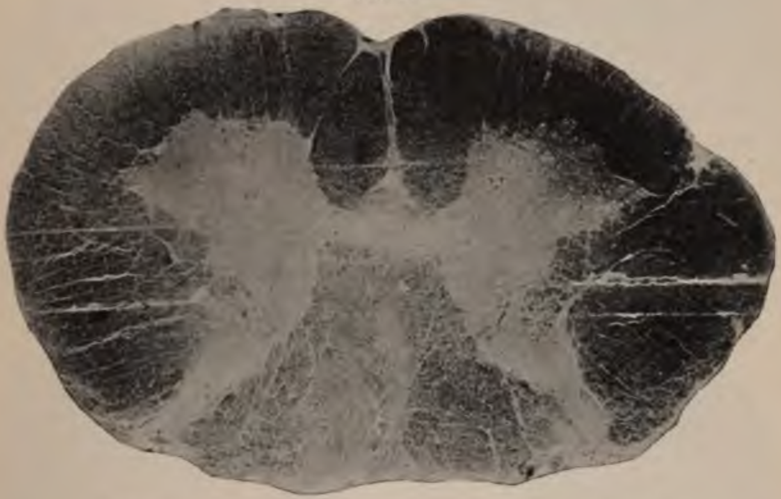


FIG. 45.

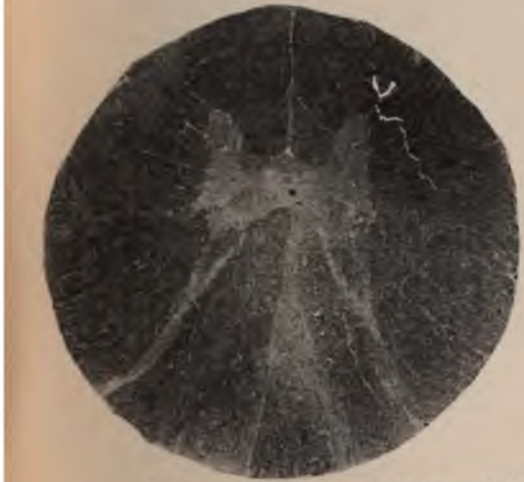
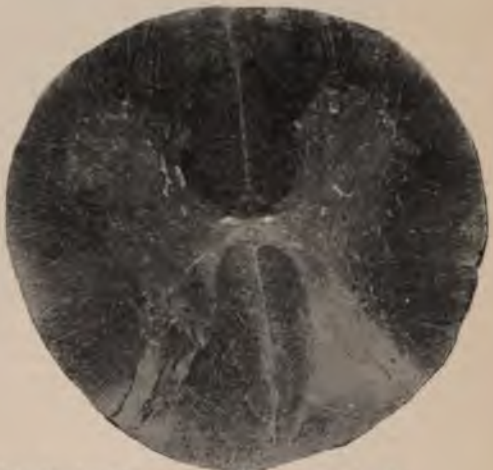


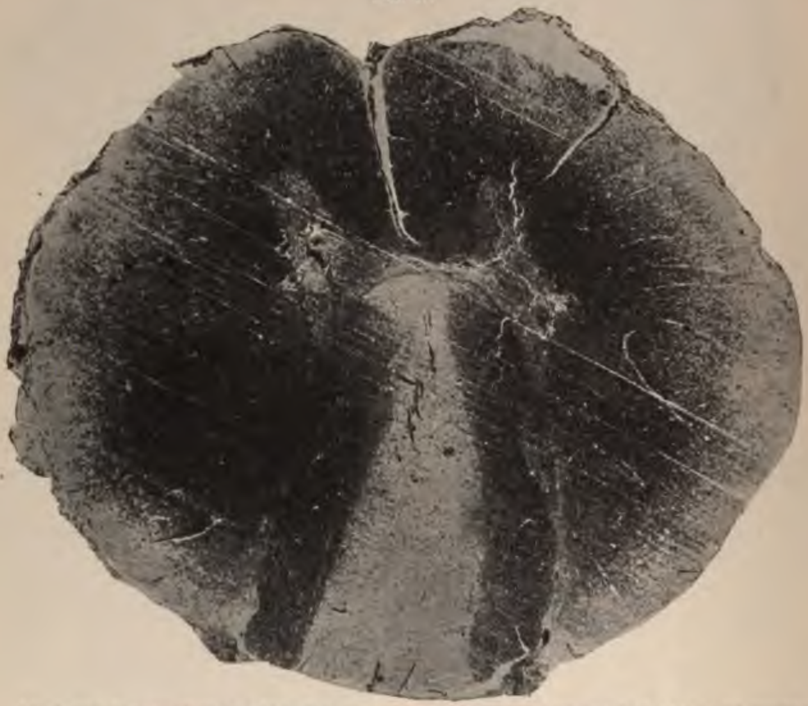
FIG. 46.



Sections of the spinal cord at the cervical (Fig. 44), dorsal (Fig. 45), lumbar (Fig. 46) levels, showing ascending degeneration, unilateral, in the posterior column after a gumma involving the second and third lumbar nerve roots. The relative extent of the degenerated fibres at the different levels is shown in the sections.

that a transverse lesion of the cord causes an ascending degeneration in the direct cerebellar column, the antero-lateral ascending tract, or column of Gowers, and many fibres of shorter or longer extent in the column of Burdach and in the antero-lateral columns. As degeneration only occurs in a fibre which is cut off from its neurone, it is evident that the neurones of which these degenerated fibres are branches lie in the cord itself and not in the posterior spinal ganglia. It has been stated already that the majority of the fibres entering a posterior root zone are short fibres. These terminate in fine brushes about the cells lying in the posterior gray matter and median gray matter of the spinal cord. Numerous neurones lie in these regions of the gray matter, and these neurones send out their axones into the lateral columns of the cord, where they turn upward toward the medulla, forming the long and short sensory columns whose existence is proven by the study of degenerations. Figs. 42 and 43 show the situation of these cells in the posterior and median gray matter of the cord with their axones passing into the various columns. In Fig. 42 it will be noticed that some cells send their fibres into the

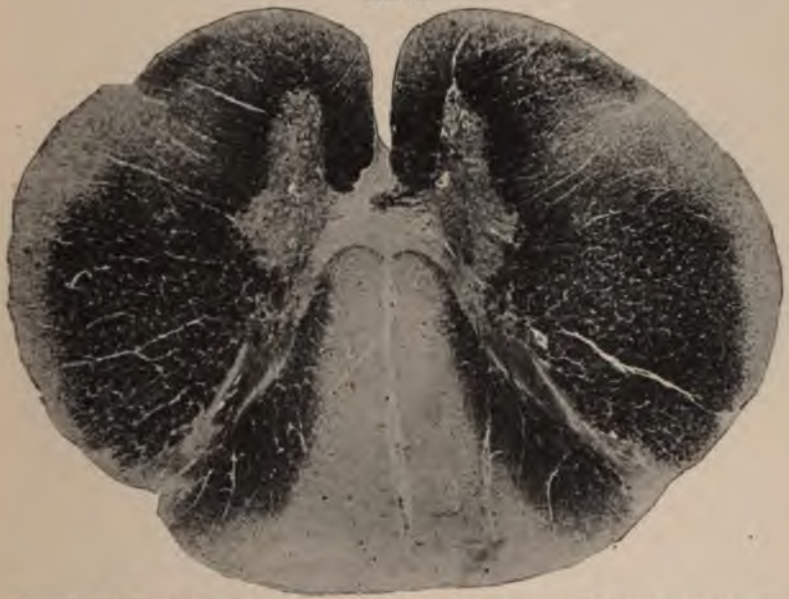
FIG. 47.



Ascending degeneration in the mid-dorsal region after transverse myelitis at the first lumbar segment. The degeneration affects the posterior median columns, together with the posterior commissural tracts, also the direct cerebellar column, the column of Gowers and many fibres in the antero-lateral columns.

columns of the cord on the side on which they lie. These are termed tautomere neurones. In Fig. 43 it will be seen that some cells send their fibres across the median line into the opposite columns of the cord. These are termed heteromere neurones. In both figures the numerous fibres from the sensory nerves entering the gray matter and terminating around these neurones are seen. It is thus evident that the course of the sensory impulses coming in through the short sensory nerve fibres is very complex. And it is evident that while some sensory impulses pass upward on the same side on which they enter, many sensory impulses are sent across the cord and pass up on the opposite side.

FIG. 48.

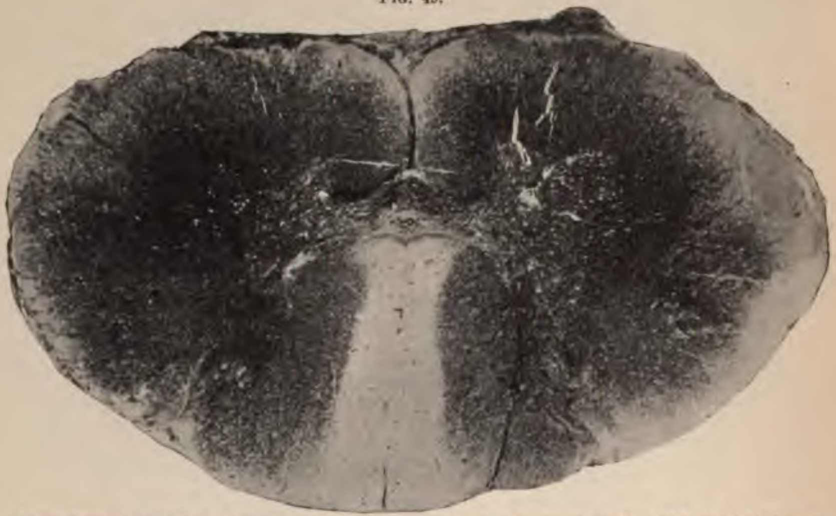


Ascending degeneration at the first dorsal segment after transverse lesion of the fifth dorsal segment. The ascending degeneration affects all of the posterior columns excepting the root zone of the column of Burdach. Also the direct cerebellar columns and columns of Gowers, and many fibres in the antero-lateral columns. A few degenerated fibres are seen around the margin of the anterior median column.

It has been already stated that sensations entering the cord are of various kinds. It is not yet possible to assign a special path to each of these various sensations, but it seems probable that the sensations of muscular sense pass upward in the long fibres of the posterior columns and in the direct cerebellar tract of the same side upon which they enter; that the sensations of temperature and of pain pass in the short fibres to the central gray matter of the side on which they enter, are then taken up by new neurones which transmit them across the cord and upward in the antero-lateral tract; that the tactile impulses enter the posterior columns and also the gray matter of the cord, and are taken up by neurones, some of which send their fibres into the columns of the same side; but the majority send their fibres across the median line into the antero-lateral columns of the opposite side (Fig. 43, *AL*). It can be positively stated that a condition of ataxia implies a lesion of the posterior column of the cord; that a condition of analgesia and thermo-analgesia implies a lesion of the central gray matter and of the ascending antero-lateral fibres, and that a condition of tactile anæsthesia implies widespread degeneration in the antero-lateral and posterior columns of the cord.

In a unilateral lesion of the cord it is usually found that there are tactile anæsthesia, analgesia, thermo-analgesia in the side of the body opposite to the lesion, together with some hyperæsthesia of all these sensations in the same side of the body as the lesion; and it is from this clinical fact that the conclusion is reached that sensory impulses pass across the cord and ascend in the columns of the opposite side.

FIG. 49.

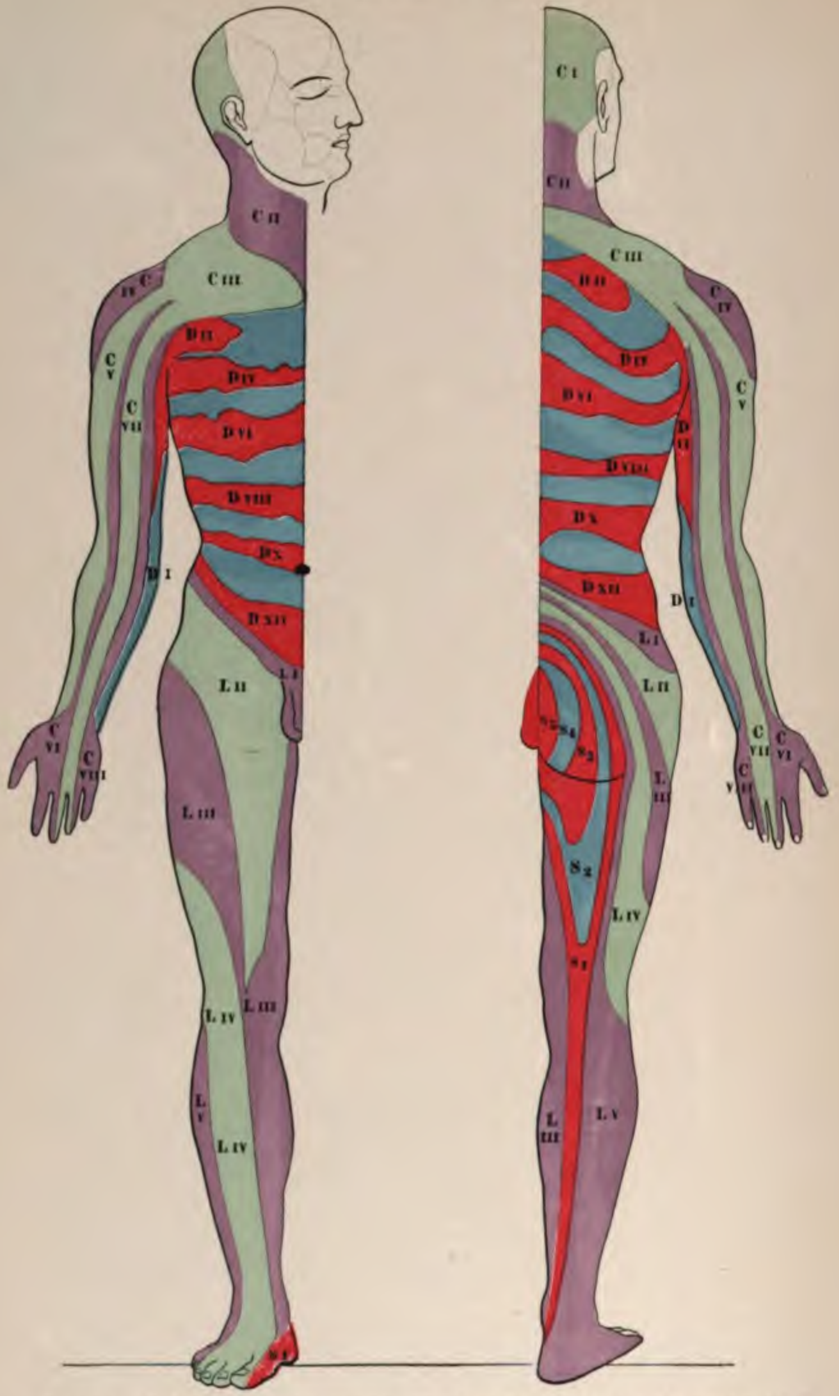


Ascending degeneration at the fifth cervical segment after transverse myelitis at the eighth dorsal segment. The columns of Goll, the post-commissural tracts, the external part of the column of Burdach, the direct cerebellar column, the column of Gowers, and some parts of the antero-lateral column are degenerated.

In syringomyelia, in which the central gray matter of the cord alone is affected there is a loss of temperature and pain sense only. This occurs on the side of the lesion. It has been supposed until recently that these sensations were transmitted upward in the column of Gowers, but recent researches prove that this column passes to the cerebellum.

In locomotor ataxia, in which the lesion is limited to the posterior root zone, all the sensations are more or less impaired, and there is secondary degeneration ascending in the posterior columns only. In general myelitis all the sensory tracts are implicated, and here too all forms of sensation are impaired.

PLATE XIII.



Areas of Anæsthesia upon the Body after Lesions in the Various Segments of the Spinal Cord.

The segments of the cord are numbered: C I to VIII, D I to XII, L I to V, S 1 to 5, and these numbers are placed on the region of the skin supplied by the *sensory* nerves of the corresponding segment.

In transverse lesions of the cord at any segment it is evident that the impulses reaching that segment from its own pair of nerves, and the impulses passing through that segment to and from the segments below it, will be cut off; hence after transverse lesions there is a condition of total anæsthesia in the body below the segment which is destroyed. If, therefore, a series of cases is brought together of lesions of the cord in every segment from the last sacral up to the upper cervical, and if the area of anæsthesia in the body in each of these cases is accurately determined, it is evident that it will be possible to ascertain the exact region of the skin related to the individual segments of the spinal cord. In Plate XIII. these areas are carefully laid down. It is evident, therefore, that in any case of spinal-cord disease it is necessary to test the sensations and to compare the area of anæsthesia with the diagram here given, and thus to determine the level of the spinal cord affected. It is to be remembered, however, that the skin of the body is plentifully supplied with sensory nerves which anastomose freely at their terminations, and the researches of Sherrington have demonstrated that each part of the skin is supplied with sensory nerves from two adjacent segments of the cord; hence a condition of anæsthesia in the skin indicates a suspension of function of two segments of the cord at least, for if one segment alone were affected the segments above and below it would be capable of supplying the skin with sensation. This conclusion, drawn by Sherrington from physiological experience, I have confirmed in a case of spinal disease in which it became necessary to divide completely the posterior nerve roots at the sixth dorsal level. This division did not produce any anæsthesia around the body, because the fifth and seventh nerves supplied the skin of the trunk in the domain of the sixth nerve sufficiently to prevent anæsthesia. If, however, two adjacent nerves are divided, a zone of anæsthesia is produced. The overlapping, therefore, of adjacent sensory areas is not to be forgotten in determining the level of the segment affected. Thus if in a case the areas of anæsthesia on the body correspond to the section shown in the diagram as belonging to the second, third, fourth, and fifth sacral segments of the cord, the lesion of the cord undoubtedly involves the first sacral segment also, but is certainly not any higher.

The determination of the area of anæsthesia is of particular importance in cases of compression of the cord by tumor or by dislocated or fractured vertebræ, as it is the most positive indication of the exact level of the cord which is invaded by disease. Thus in the cases in which tumors have been successfully removed from the cord the level of the anæsthesia has been the guiding symptom for the surgeon. In such cases the normal anatomical relation between the segments of the spinal cord and the vertebræ is not to be forgotten.

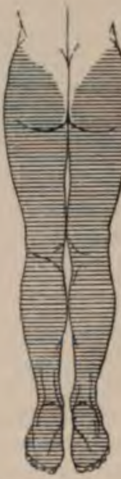
Small areas of anæsthesia in the body corresponding in distribution to the diagram, and due to small localized foci of disease in the spinal cord, are found chiefly in syringomyelia, in hemorrhages in the cord, in small areas of softening in the cord, or in tumor of the cord. These lesions destroy the terminal filaments of two or three sensory nerves and do not invade the long tracts coming from the parts below the level of the lesion; hence localized anæsthesia is indicative of a small limited lesion, not of general transverse diseases. Such small limited lesions are not very common. In all cases the anæsthetic area fades gradually into the area of normal sensibility. In hysteria the line of demarcation between sensitive and insensitive skin is much sharper.

The determination of the exact area of anæsthesia in any case is of great service also in differentiating lesions of the spinal cord—first, from lesions of the nerve trunks; secondly, from lesions of the cauda equina; thirdly, from hysteria; fourthly, from multiple neuritis. In lesions of the nerve trunks the distribution of the anæsthesia is different from its distribution in the spinal-cord affections. This will be evident if the areas in Plate V. showing the distribution of the nerves of the skin is compared with the areas in Plate XIII. showing the distribution of the nerves from the segments.

Fractures of the lower lumbar vertebræ or of the sacrum often produce pressure upon the cauda equina. It has been found that when such pressure is slight, it is the nerves lying innermost within the cauda which suffer most, and the greater the compression the greater the number of nerves which suffer. The distribution of the anæsthesia in caudal lesions resembles closely the distribution of anæsthesia in lesions of segments of the cord, and from a study of the anæsthesia alone it is impossible to differentiate absolutely a caudal lesion from a cord lesion. A diagnosis may, however, be made—first, from a study of the surgical indications, chiefly of the nature of deformity, the relation of the vertebræ to the segments of the cord being remembered. The spinal cord ends at the first lumbar vertebra; hence any fracture below that level necessarily compresses the cauda equina and does not destroy the spinal cord. Secondly, a diagnosis may be made from a study of the paralysis which accompanies the anæsthesia. This paralysis is very slight in lesions of the sacral segments of the cord. Thus when a lesion is at or below the third sacral segment the paralysis is confined to the peronei muscles. When the first sacral segment is also involved, the paralysis affects the anterior and posterior tibial muscles, and it is only when the entire lumbar region of the cord is destroyed that movements of the hip-joint will be affected. In cauda-equina lesions, on the other hand, pressure upon the nerve roots is often sufficient to produce widespread paralysis when sensation is but slightly affected. Thus in a lesion of the spinal cord the distribution of the paralysis will correspond to the segment of the cord invaded by disease, and will correspond to the distribution of the anæsthesia produced by a lesion of that segment, while in lesions of the cauda equina the distribution of the paralysis may be much more extensive than that indicated by the distribution of the anæsthesia.

The distribution of the anæsthesia is also of much service in differentiating hysterical paralysis from organic disease of the spinal cord. Charcot pointed out the fact that in hysterical paraplegia the anæsthesia never involves the genital organs or the perineum and the anus. Paralysis of the bladder and rectum is a very rare thing, and if it occur is of the nature of retention of urine rather than of incontinence. Fig. 50 shows the area of anæsthesia commonly observed in hysterical cases, and it will be noticed that its distribution is very different in outline from that in Plate XIII. It is also to be remembered that in hysterical paraplegia there is no reaction of degeneration in the muscles, and there is no loss of tendon reflexes.

FIG. 50.



The area of anæsthesia in hysterical paraplegia; the genitals are not anæsthetic.

In many cases of traumatic neurosis or of irritation of the spine following injuries, and in the so-called spinal concussion associated with anæsthesia, it will be found that the distribution of the anæsthesia corresponds to the hysterical rather than to the organic type.

In multiple neuritis the region of anæsthesia assumes a stocking-shaped or glove-shaped area on both limbs symmetrically, and does not extend to the trunk; already shown in Fig. 27.

It is important to be able to locate the lesion accurately in spinal-cord disease, no matter whether the lesion thus determined corresponds to well-known forms of spinal disease or not. It has recently been shown[1] that vascular diseases of the spinal cord, hemorrhages into the cord of small extent, or long perforating hemorrhages in the cord of small lateral extent, or small areas of softening of the cord due to thrombosis of diseased spinal arteries or their branches, are more common than was supposed. The diagnosis of these conditions is only possible by an accurate study of symptoms, and such a study may indicate a local lesion suspending the function of a small area at any level.

Ataxia. Ataxia is a symptom of spinal-cord disease due to an interference with the muscular sense impressions which pass into the cord through the posterior nerve roots and root zone. Both the automatic and voluntary movements of the body are guided by the impressions received through muscular sense, and if those impressions are lacking the movements become awkward. Such movements can be guided by the eye, and hence an ataxic will always perform a motion more deftly if he watches the limb which is moved, but deprive him of vision by closing his eyes and the symptom of ataxia is readily developed. The lesions of the spinal cord which

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[1] R. A. Williamson. Manchester Medical Chronicle, 1895.

produce ataxia are those which destroy the fibres in the posterior external column or column of Burdach, through which the fibres pass which convey the impressions of muscular sense. As has been already stated, these fibres pass upward and downward in the column of Burdach, sending their collaterals into the posterior gray matter of the cord at different levels, so that the impressions coming in through a single nerve are conveyed to many segments of the cord. In addition, there are the long tracts passing to the medulla, already described in the column of Goll. When one thinks of the very numerous and complex movements which are involved in such a simple act as lifting a glass of water to the lips, involving almost the entire body, made in order to preserve its balance, one realizes that to properly guide any movement, however simple, a very extensive action of a large number of muscles is required. This action is guided automatically by the muscular sense. It is only when the muscular sense is interfered with that one realizes its extensive use and its constant function. The disease in which ataxia is most evident is posterior sclerosis, but any lesion affecting the same area of the cord will produce ataxia. Thus multiple sclerosis, or tumors of the meninges on the posterior surface, or tumors within the cord, or syringomyelia when it involves the posterior columns, or a general myelitis, are capable of producing this symptom. The ataxic gait has already been described.

Pain. Pain is a symptom of considerable importance in spinal-cord disease. It may be felt in the spine itself; that is, in the back and deeper structures, under which circumstances, as a rule, there is a more or less extensive affection of the nerve roots or of the meninges, but not of the spinal cord. Severe pain in the back and spinal ligaments is not at all uncommon in functional affections, such as traumatic hysteria and traumatic neurasthenia; in nervous prostration; in hysteria. It is sometimes present in the affections of the viscera, under which circumstance the pain is really a "referred pain," referred to the back when the actual irritation comes from the sympathetic nervous system connected with the viscus diseased. (See page 99.) In organic affections of the bones, ligaments, and nerve roots spinal pain is also a frequent symptom.

When pain is produced by disease of the spinal cord itself, it is due to an irritation or injury of the posterior nerve roots at their entrance or to an irritation of the sensory tracts passing upward through the spinal cord. Under these circumstances the pain is not referred to the back, but is felt in the part of the body from which the irritated nerve root or sensory tract has come. Thus in locomotor ataxia the sharp shooting pains are referred to the limbs rather than to the back, and as the disease almost uniformly begins in the second and third lumbar segments of the cord, these pains are usually referred to the anterior surface of the thighs, and as the disease advances downward to the fourth and fifth lumbar segments, the pain is felt in the feet; as it advances upward into the dorsal region, the pain is felt about the body. When the process has ascended to the lower cervical region pains begin to be felt in the axilla, on the inner side of the arms, and in the little fingers, and finally, as the upper segments of the cervical region are invaded by the sclerotic process, the entire arms and shoulders become the seat of pain. The location of pain, therefore, in any case of disease of the cord is an indication of the level of the disease, and comparison of the distribution of the pain in any one case with the diagram (Plate XIII.) will enable one to locate the affection.

In syringomyelia, in which the terminal filaments of the pain-sense nerves within the gray matter are destroyed, the analgesia which finally develops is usually preceded by a stage of pain, and here too, as in locomotor ataxia, the pain is referred to the periphery corresponding to the segments of the cord involved.

Pain referred to the extremities is also a symptom in injuries of the cord, in hemorrhages within the cord, or in crushing of the cord such as follows dislocation or fracture of the spine. In these cases the pain is referred to the periphery, but is less exactly referred than in locomotor ataxia, for the injury affects all the sensory tracts coming from the parts below the site of the lesion, and these are damaged to a greater or less extent; consequently, the reference of the pain is more widespread. Thus in a hemorrhage in the lower cervical region intense pain may be felt in the entire body below the level of the arms. In these cases any movement of the body causing an increased mechanical pressure upon the cord may be attended by sharp, shooting pains felt in any part of the body below the lesion. Pain is an early symptom in tumors of the cord, and in these cases it may be referred to the periphery from which the nerve root comes, which is primarily compressed at the site of the tumor. Thus in a tumor in the dorsal region the pain may be felt in the thorax, epigastrium, or abdomen. It may be also referred to the parts of the body below the tumor, because of the irritation of the sensory tracts passing through the cord at the site of the tumor.

There are some cases in which pain is referred to an anæsthetic portion of the body. Thus in caries of the spine, in localized meningitis, in injuries of the nerve roots, and in some cases of sclerosis of the cord the sensory nerves may be destroyed, so that no sensations can pass in over them, and hence the surface of the body may be anæsthetic; but at the same time the proximal ends of these destroyed nerves may be irritated by the disease, and hence painful sensations may constantly be sent inward to the cord. These, being received and transmitted upward, are referred to the anæsthetic surface, giving rise to the symptom called anæsthesia dolorosa. This condition is very rarely seen in diseases of the cord itself, but is very common in diseases of the vertebræ, especially in tumors (carcinoma, sarcoma) and in caries of the spine.

Vasomotor and Trophic Symptoms. In the gray matter of the cord there lie certain cells which regulate the mechanism by which nutrition in the body is maintained. It seems probable that these mechanisms are set in activity by sensory impulses reaching the cord through the posterior nerves, because many posterior nerve or nerve-root lesions are attended by trophic disturbances—*e. g.*, joint disease and perforating ulcer of the foot in locomotor ataxia. In locomotor ataxia the joint diseases are quite frequent, the ankle, knee, and hip being affected in the order mentioned. In syringomyelia it is the elbow, wrist, fingers, and shoulder, in the order mentioned, which are most commonly affected. It is quite noticeable that such joint affections occur chiefly among the lower classes or in persons who are exposed to injury or to falls; hence it is concluded that their origin is traumatic, even a slight injury being sufficient to produce them. There is usually at first an effusion of fluid into the joint, subsequently a thickening of the ends of the bones. If such joints are treated by perfect rest at an early stage, the disease disappears. If, however, they are neglected, as they are very liable to be because of the absence of pain, the process goes on and increases until the joint is rendered useless. It is a very suggestive fact that these joint diseases appear only in those spinal affections in which the sense of pain is impaired, and this is another proof of the conservative influence of pain in securing rest or immobility in the diseased part.

The mechanisms started by these sensory impulses which control nutrition lie in the central gray matter of the cord, and to some extent in the anterior horn, for trophic disturbances, especially eruptions in the skin, necrosis of the fingers, atrophy of the muscles, fragility of the bones, occur in lesions limited to the gray matter of the central area or invading the anterior horn, as, for example, in syringomyelia and in anterior poliomyelitis. It is probable that these mechanisms are not the only ones which preside over nutrition, and that throughout the body and in the skin the nerve mechanism of the vessels is quite competent to regulate nutrition, provided it is not put to too great a strain. Thus bed-sores and cystitis are in my opinion rarely due primarily to lesions of the spinal cord, and may usually be avoided by proper care. In the normal body continuous pressure upon any one part for a long time without shifting of position does not occur even in sleep, and if a similar repeated change of posture is kept up in a case of spinal paralysis, bed-sores will not appear. It is undoubtedly the continued pressure without change of position which produces such sores, and this is proven by the locality in which they uniformly appear—namely, over the sacrum, upon the trochanters, upon the heels, or upon spots subjected to greatest pressure when the patient lies long in one position. In cystitis it is the introduction of germs into the bladder by means of the catheter which is responsible for the disease, or else an over-distention and consequent inflammation of the bladder when the proper catheterization is neglected. While it is admitted, therefore, that the spinal cord has trophic functions in the sense that it regulates the degree of circulation and the rapidity of processes of nutrition, it is not believed that a lesion in the cord alone under proper care of the patient will result in lesions of the skin or mucous membranes.

The atrophy of the muscles which occurs in spinal paralysis appears to be too rapid to be accounted for by disuse, and the fragility of the bones which appears in syringomyelia can only be accounted for by a distinct trophic influence of the cord upon the bones.

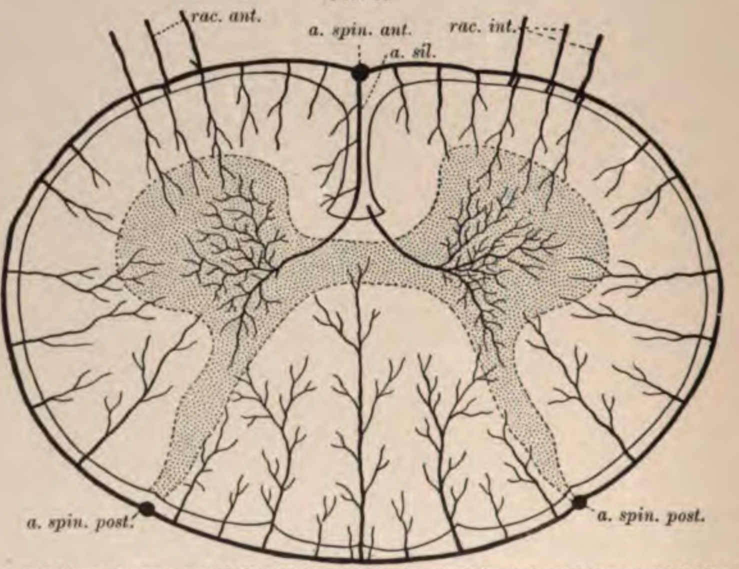
In all conditions of destruction of the spinal cord the part of the body related to the part of the cord destroyed is found to present a bluish appearance, indicating an imperfect capillary circulation due to a lack of tone in the arteries, and also a cold condition due to imperfect processes of nutrition going on in consequence of the venous stasis. This is particularly noticeable in the parts of the body below a transverse lesion of the cord, in syringomyelia, and also in anterior poliomyelitis; hence it is evident that the spinal cord is concerned in the vasomotor mechanism. Hence in any cases where vasomotor or trophic symptoms appear in a patient the suspicion is aroused of a spinal-cord disease.

CHAPTER XI.

THE SPINAL BLOODVESSELS.

THE main arteries of the spinal cord are three in number. They lie on the anterior and posterior surfaces of the organ along its entire length. The anterior spinal artery is formed by the junction of two vessels which arise from the vertebral arteries and it extends to the lowest part of the spinal cord. The posterior spinal arteries also arise from the vertebral arteries, but do not often join. They pass downward along the surface of the cord on each side near the entry of the posterior spinal nerve roots. (See Plate XI.) In addition to these main arteries there are a large number of smaller arteries, branches of the intercostal arteries, which enter the spinal canal at the sides of the spinal nerves and are distributed to the antero-lateral surfaces of the cord, freely anastomosing with one another. These have been called anterior and posterior radicular arteries. They divide into ascending and descending branches and anastomose freely with the anterior and posterior spinal arteries, making a sort

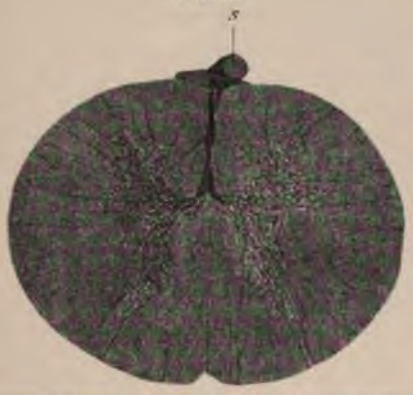
FIG. 51.



Scheme to show the course and distribution of the terminal branches of the arterial plexus of the pia mater. *a. spin. post.*, posterior spinal arteries; *a. spin. ant.*, anterior spinal arteries; *a. sil.*, anterior median fissure; *rac. ant.*, anterior root arteries. (After Van Gehuchten.)

of arterial network about the spinal cord from which numerous small branches penetrate the surface of the organ. The number of these radicular arteries is variable, but they are never present on all the nerves, every second or third nerve having an accompanying vessel. There is a fold of pia mater in the anterior fissure of the spinal cord, and in this fold a large branch of the anterior spinal arteries is usually found running longitudinally. From this branch secondary branches enter the gray matter of the spinal cord and pass down in it, forming the central arteries of the cord. Thus the supply of the gray matter and white matter of the cord is from different branches. Fig. 52 demonstrates this distribution. All of the spinal arteries which enter the cord are terminal arteries like the basal arteries of the brain; that is, they do not anastomose. Hence an embolus in a spinal vessel always leads to an area of softening. The peripheral spinal branches which penetrate the organ from its surface enter with the connective-tissue septa or with the nerve roots. They supply the white matter of the cord, and their terminal twigs reach the edge of the gray matter and sometimes enter it, but they do not anastomose with the branches of the central artery.

FIG. 52.



The distribution of the anterior spinal artery and vein to the gray matter of the spinal cord. An injected preparation. (Adamkiewicz.)

The fine capillaries within the gray matter empty into veins which accompany the central arteries and empty into larger veins which lie in the anterior fissure. The majority of the spinal capillaries, however, empty into fine venous twigs which make their exit on the lateral and posterior surfaces of the cord where a venous network surrounds the cord. This in turn empties into larger veins which make their exit from the spinal canal with the spinal nerves, and thus reach the vena cava. Others ascend to empty into the branches of the jugular vein within the skull.

While little attention has hitherto been paid to diseases of the spinal bloodvessels and to the results of endarteritis, it is probable that these play a large part in the production of various forms of spinal-cord disease. Since the discovery of the characteristic symptoms of syringomyelia it has been possible to diagnosticate hemorrhages within the gray matter of the cord by the sudden appearance of similar symptoms; and recent pathological study[1] seems to indicate that many supposed cases of myelitis are really cases of softening in the cord due to thrombosis in diseased bloodvessels, and that some forms of sclerosis may originate in obliterating endarteritis of the peripheral vessels that enter the posterior and lateral columns of the spinal cord. It will be necessary in the chapters upon tabes and upon lateral sclerosis to call attention more particularly to endarteritis as a possible cause of some cases of these diseases.

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[1] R. J. Williamson. Manchester Medical Chronicle, 1895.

CHAPTER XII.

ANTERIOR POLIOMYELITIS.

Acute Anterior Poliomyelitis. Infantile Paralysis. Chronic Anterior Poliomyelitis. Progressive Muscular Atrophy. Ascending Atrophic Paralysis.

ANTERIOR poliomyelitis, infantile spinal paralysis, acute atrophic paralysis, atrophic spinal paralysis, or regressive paralysis is an acute disease, chiefly observed among children, but occasionally among adults, characterized by sudden complete loss of power in one or more limbs, usually in the legs, followed by rapid atrophy of the paralyzed muscles and by an imperfect growth of the limb affected, and attended by slight pain, but not by any permanent sensory disorder.

Etiology. The disease occurs in both sexes with about equal frequency. There is no history of its being inherited. The following table demonstrates that the age of maximum liability is between the first and fourth years, but children at all ages are liable to the disease, and it occurs in adult life:

TABLE IV.—*Age of Onset.*

	1st year.	*2d.*	*3d.*	*4th.*	*5th.*	*6th.*	*7th.*	*8th.*	*9th.*	*10th.*
Seeligmüller,	20	25	18	1	1	2				
Galbraith,	17	88	15	4	1					
Sinkler,	44	92	55	29	9	2	3	6	0	3
Gowers,	21	21	25	9	17	4	2	6	4	0
Starr,	19	52	45	23	18	8	5	2	5	4
Total cases,	121	228	158	66	46	16	10	14	9	7

The youngest case on record is mentioned by Duchenne in a child twelve days old, and Sinkler has seen a case develop in a child at the age of six weeks. The youngest patient in my own records was five months old. It has been noticed by all authors since the time of Barlow (1878) that infantile paralysis develops most commonly during the warm season. This is especially true in England and in America, as is shown by Table V., which demonstrates the month of the year in which cases developed, as noted by Barlow, Gowers, Sinkler, and myself:

The disease has occurred in epidemic form in a number of different localities in every case during the summer. Colmer[1] first recorded the occurrence of the disease in epidemic form, for he mentions that in a village where he saw 1 case 10 other cases had developed during the preceding few weeks. Cordier[2] published an account of an epidemic occurring in Lyons, France, in 1885. He saw 13 cases developing during the months of June and July in a small town of 1500 people where in other years the disease had been extremely rare. Medin of Stockholm described an epidemic of the disease occurring in the months of August, September, and October, 1889, 44 cases having been observed by him during that time; and Rissler, who examined 3 of these cases post-mortem, demonstrated that it was a true anterior poliomyelitis. Medin mentions that a small epidemic had occurred in 1881 in the town of Umea in Sweden. Leegard observed a small epidemic in Mundal in Norway in 1890.

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[1] American Journal of the Medical Sciences, 1843.

[2] Lyon médical, 1887.

TABLE V.—*Month of Onset.*

	Barlow.	*Gowers.*	*Sinkler.*	*Starr.*	*Total.*
January	1	1	4	3	9
February	0	1	3	1	5
March	4	1	9	7	21
April	2	1	4	3	10
May	4	1	10	4	19
June	5	11	27	7	50
July	16	13	52	30	111
August	11	13	65	48	137
September	4	15	29	34	82
October	3	6	25	13	47
November	1	2	4	5	12
December	2	5	3	2	12
	53	70	235	157	515

The most extensive epidemic of the disease on record was described by Caverly[1] of Rutland, Vt. It occurred between July 20 and September 20, 1895, in the Otter Creek Valley, within a radius of twelve miles of the city of Rutland, and during the summer an unusual number of isolated cases of the disease were observed through the State of Vermont. Caverly reports 144 cases of various grades of severity developing both in children and adults, children below the age of six years being the chief victims.[1] Pieraccini[2] observed a small epidemic near Florence, Italy, in the same year (1895) in July and August; and Medin[3] of Stockholm has recently reported a second epidemic in Stockholm in the year 1895. The occurrence of the disease in epidemic form is exceedingly suggestive of its infectious origin. In confirmation of this hypothesis it is to be noted that the disease has been frequently observed in connection with other infectious diseases. Thus it is an occasional sequel of diphtheria, meningitis, measles, pneumonia, scarlet fever, and acute malarial poisoning

Exposure to cold or to a sudden check of perspiration has been supposed to be an exciting cause in certain cases. I have seen several children who were attacked with infantile spinal paralysis subsequent to long-continued bathing in cold water during the summer. Traumatism is frequently assigned as a cause by parents, and a few cases are on record, which I can conform by my own observations, in which the disease has developed immediately after a severe fall or blow on the back. In some cases no cause can be discovered.

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[1] New York Medical Record, December 1, 1885.

[2] Lo Sperimentale, xlix., No. 27, September, 1895.

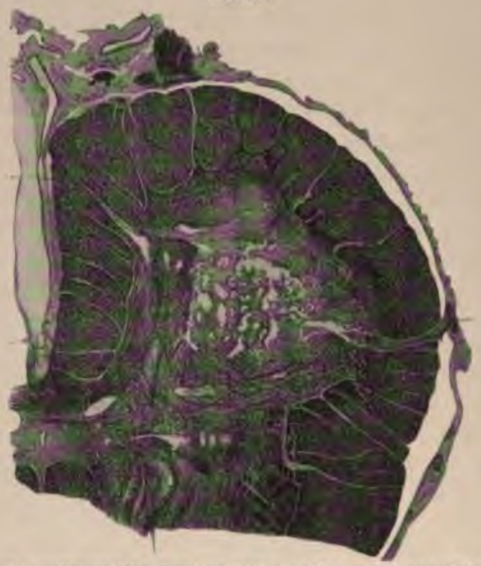
[3] Nord. Med. Ark., 1896, No. 1.

The frequency with which the disease appears in children who are learning to walk, together with the fact that the symptoms are located much more commonly in the legs than in the arms, has led to the supposition that a functional hyperæmia of the cord in its lumbar region, due to overexertion, going on to a pathological congestion and hemorrhage, may be an etiological factor.

Pathological Anatomy. It is only within the past few years that the exact pathological changes occurring in infantile spinal paralysis have been accurately described, autopsies having been recently obtained within a few days of the onset of the disease and at longer intervals in different cases up to the state of chronic permanent change in the cord. The earlier description of Charcot was based upon cases examined only in the chronic stage of the disease, and this fact explains the discrepancy between his observations and those of modern pathologists.

In the early stage of the disease there is active congestion of the spinal meninges and of the gray matter of the spinal cord supplied by the branches of the anterior spinal artery. The bloodvessels are distended and some of the capillaries are ruptured, allowing extravasations of blood cells; the perivascular spaces and the gray matter of the cord are filled with emigrating leucocytes; and there is a considerable exudation of serum. The serum fills the lymph spaces about the vessels and about the nerve cells; the leucocytes infiltrate the tissues everywhere, cluster about the cells, and make their way into the cells. There is a great increase of small cells and nuclei throughout the neuroglia, which may be due to a proliferation of the neuroglia cells or of the endothelial elements, or may be due to an emigration from the bloodvessels. This infiltration of the tissues with leucocytes and nuclei may be so intense as to obscure all other elements. It is thus evident that the bloodvessels and the neuroglia, as well as the ganglion cells in the gray matter of the cord, share in the pathological process. The changes in the motor neurones (ganglion cells) are very marked. All varieties of degenerative changes may be seen. The cell may have a cloudy appearance and be slightly swollen, staining more deeply by reagents, the chromophile granules appearing to be larger than in the normal cells, as seen by the Nissl stain, and the nucleus appearing granular. This is shown in Plate III., B. A further stage of degeneration is shown by the fact that the protoplasm no longer absorbs stains; the cell is swollen, has lost its sharp outline, has a homogeneous appearance, and the nucleus is faint, as are also the outlines of some of the dendrites. It is probable that in both these stages of degeneration an arrest of the process and a gradual regeneration and a return to the condition of health with resumption of the function of the cell is possible. If the process of degeneration proceeds beyond this point, however, no repair is possible, and the function of the cell is forever lost. When degeneration has gone on beyond the stage last described the cell appears to be changed into a swollen, irregular, or spherical mass of material; its protoplasm becomes cloudy and its nucleus is not visible, if stained at all; the chromophile granules have lost their regular arrangement in concentric rings about the nucleus or in radiating lines toward a neuraxone, and the homogeneous mass is seen to be permeated with vacuoles, the dendrites having dropped off. (See Plate III., C., D.) In the last stage the protoplasm shrinks, the cell body being reduced very materially in size. It has lost its polygonal shape and become no larger than its original nucleus. It stains deeply and has a granular appearance. During the later stages of this degeneration leucocytes may be observed penetrating the pericellular spaces and encroaching upon the cell body. Both in the dendrites and in the neuraxone similar degenerative processes may be observed in progress, and they are destroyed before the cell undergoes its final degeneration. (See Plate III., E., F., G.)

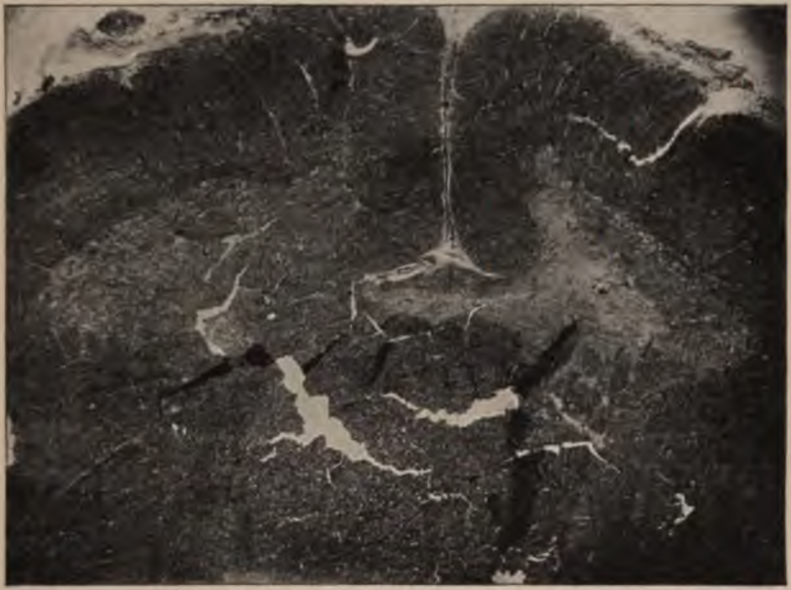
FIG. 53.



Hemorrhagic anterior poliomyelitis, lumbar region. The substance of the anterior horn has been disintegrated by the hemorrhage, and all the cells destroyed. (Siemerling, Arch. f. Psych., xxvi., p. 290.)

While in the majority of cases there is a parallel degree of change in the interstitial tissues and in the ganglion cells, so that there is a shrinkage and progressive destruction of the neuroglia as well as of the nerve elements, there are a few cases in which the cellular degeneration is attended by few changes in the interstitial tissues. The degree to which various groups of cells are affected varies greatly at different levels of the cord, and in some groups a larger number of cells may be affected than in others. The extent of the paralysis and the degree of the paralysis in any one muscle will necessarily depend upon the number of groups of cells affected and upon the number of cells destroyed in any one group.

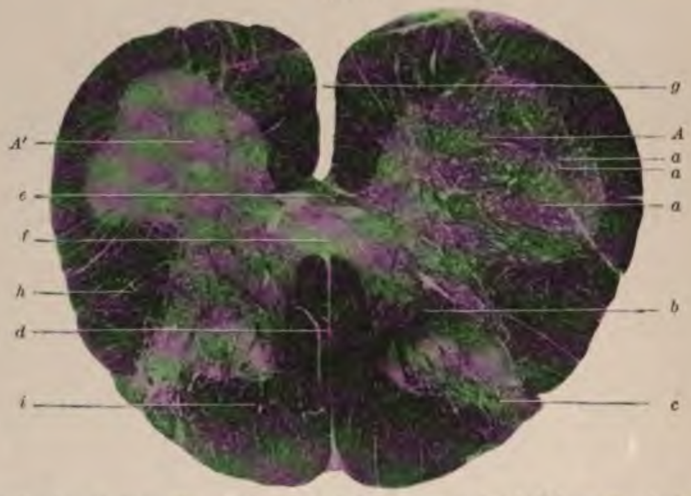
FIG. 54.



Spinal cord at sixth cervical level, from a case of infantile paralysis. The atrophy of the right anterior horn, the existence of sclerotic scar tissue in the horn, as well as the absence of groups or cells, are shown. The left anterior horn is normal.

The result of the atrophy of cells and of the neuroglia is a gradual shrinkage of the entire area of the anterior horn, leading secondarily to a collapse inward of the white columns surrounding the gray matter and of the nerves fibre issuing from the horn through these white columns into the anterior nerve roots. There is also a degenerative atrophy of fibres in the anterior nerves. Many of the cells in the anterior horns of the cord send their axones to the antero-lateral column, where they turn upward and downward to pass to other levels and terminate in the anterior horn, thus serving to associate the action of different cells lying at different levels of the cord. These association cells as well as the motor cells suffer from degeneration, and hence there is a secondary degenerative process in their axones, leading to a shrinkage and slight sclerosis in the antero-lateral column of the cord for a varying distance above and below the seat of the lesion. The extent of the lesion in the gray matter varies in various cases. In some cases the pathological change is strictly limited to the anterior horn, being particularly severe in its peripheral region. In other cases the central gray matter which lies between the anterior and posterior horns is also affected. It is in these latter cases that the symptom of pain is marked in the early stage of the disease, and the growth of the limbs is interfered with in the chronic stage, the central gray matter of the cord having a closer relation to the growth of the tissues than other parts.

FIG. 55.



The lesion in anterior poliomyelitis on the left side; entire left half is small. *A*, right side, normal anterior horn, with groups of cells (*a*); *A'*, left side, anterior horn without cells, in state of cicatrization; *b*, posterior horn; *c*, posterior nerve root; *d*, posterior septum; *e*, anterior commissure; *f*, posterior commissure; *g*, anterior fissure; *h*, pyramidal tract; *i*, posterior column. (Blocq.)

The majority of recent pathologists believe that in anterior poliomyelitis there is an acute inflammatory process limited to the domain of the anterior spinal arteries, involving both the neuroglia and the ganglion cells, and resulting in degeneration and atrophy both of the interstitial tissue and of the ganglion cells. A few observers, among whom Von Kahlden[1] may be cited, still believe that Charcot was right in supposing that the degeneration is limited exclusively to the cells, and is not accompanied by any general inflammatory process in the interstitial tissues. In a number of cases the explanation of the origin of the process is found in a thrombosis of one of the branches of the spinal artery or in a hemorrhage into the anterior horn.

It is probable that in the cases that are due to infection the lesion is one of acute inflammation, such as we find in other organs in acute inflammatory infectious diseases—*e. g.*, the lung in pneumonia, the joints in rheumatism. It is probable that in the cases in which there is an onset without fever or evidence of an infectious process the lesion is a primary degeneration of the ganglion cells or is due to hemorrhage or to thrombosis in a spinal vessel.

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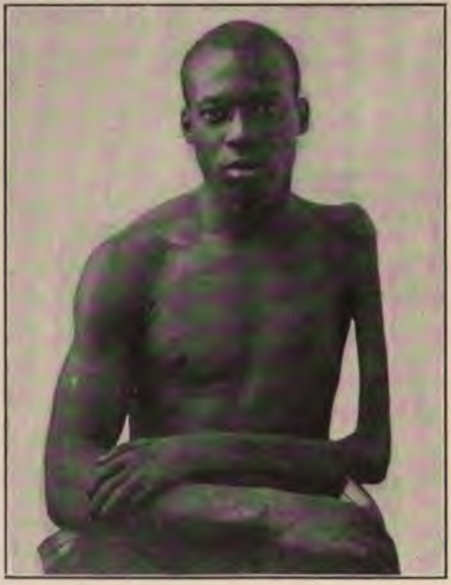
[1] Centralblatt f. allgem. Path. u. Path. Anat., 1894, vol. v. p. 729.

Symptoms. The disease usually begins, like an acute infectious disease, with fever, sometimes attended by convulsions and delirium, sometimes by considerable pain in the back, body, and limbs; occasionally by digestive disturbances, vomiting, and diarrhœa; sometimes merely by general malaise. The temperature rises rapidly to 102° or 103° F., and the patient may have a chill followed by sweating. The temperature remains about 101° or 102° for several days, with slight morning remission, then gradually sinks to normal, the entire febrile movement rarely lasting more than a week. Within a day or two of onset paralysis sets in. This may be in both legs, or in both arms; rarely in one limb alone, or in all four extremities. If the child is young and is confined to bed by the fever, the paralysis may not be noticed until the second or third day. In older children and adults the paralysis is well developed within twenty-four hours of the onset. It is observed that infants cry a good deal during the period of invasion, and those who are old enough to complain say that they suffer from pain in the back and in the affected limbs. This pain may remain for some weeks. Occasionally there is some rigidity of the spine or neck, suggestive of meningitis, but this soon subsides. There is usually no disturbance of the bladder or rectum, although in a few cases retention of urine has been noticed for a few days. There is no tendency to the development of bed-sores or of trophic changes in the skin. There is no complaint of numbness or of paræsthesia, and there is never any loss of sensation, but the limbs are sometimes painful upon any movement, especially in the joints.

After the fever with its attendant malaise and digestive disturbances has subsided and the general health has been restored, there remains a paralysis more or less extensive. This paralysis is usually more extensive at the onset than it is destined to be permanently. Thus the child may at first be completely helpless, and later recover power in all but one limb; or the trunk may be paralyzed at the onset, but not permanently affected. Both legs are commonly affected together, but the final paralysis is usually limited to one limb. Occasionally the neck muscles are distinctly weak, and there may be difficulty in swallowing. This is seen in cases in which the arms are paralyzed, and yet the final paralysis may affect but one arm. The face has been paralyzed with the arms, and the ocular muscles also, but either is a rare occurrence. In a number of cases in which the final paralysis has been limited to two or three muscles the original paralysis was widespread, involving all the limbs. The facts should be remembered in giving a prognosis in the early stage. Sometimes the onset of the paralysis is not sudden, but there is a gradual increase during a week or ten days, then a stationary period, and then a regression. The subsidence of the paralysis begins from a week to two months after the onset, and then goes on steadily, but it is not until after three months that it is quite possible to determine what muscles will eventually recover. There is always a certain amount of permanent paralysis.

The muscles which are paralyzed undergo atrophy. This is more rapid and complete in those that are to be permanently paralyzed, and there is a change in the size of the limbs which is well marked within a month. The paralyzed muscles are relaxed, never rigid, and show a reaction of degeneration to the electrical tests. The reaction of degeneration consists of a loss of the response in both muscle and nerve to faradic stimulus, and a loss of response in the nerve to galvanic stimulus. The galvanic reaction of the muscle remains, but in such a muscle there is found an alteration of its normal contractility to galvanic currents. For the first few months the muscle responds too strongly to galvanism and contracts under the positive pole more quickly than under the negative pole when the current is sent through it. Later the contractility to galvanism is progressively decreased, until in a totally paralyzed muscle it is lost. It may be stated as a prognostic sign that the muscles in which the faradic reaction is preserved will recover though paralyzed for a time at the onset. Such muscles also preserve their tone, so that they contract when percussed sharply with a hammer.

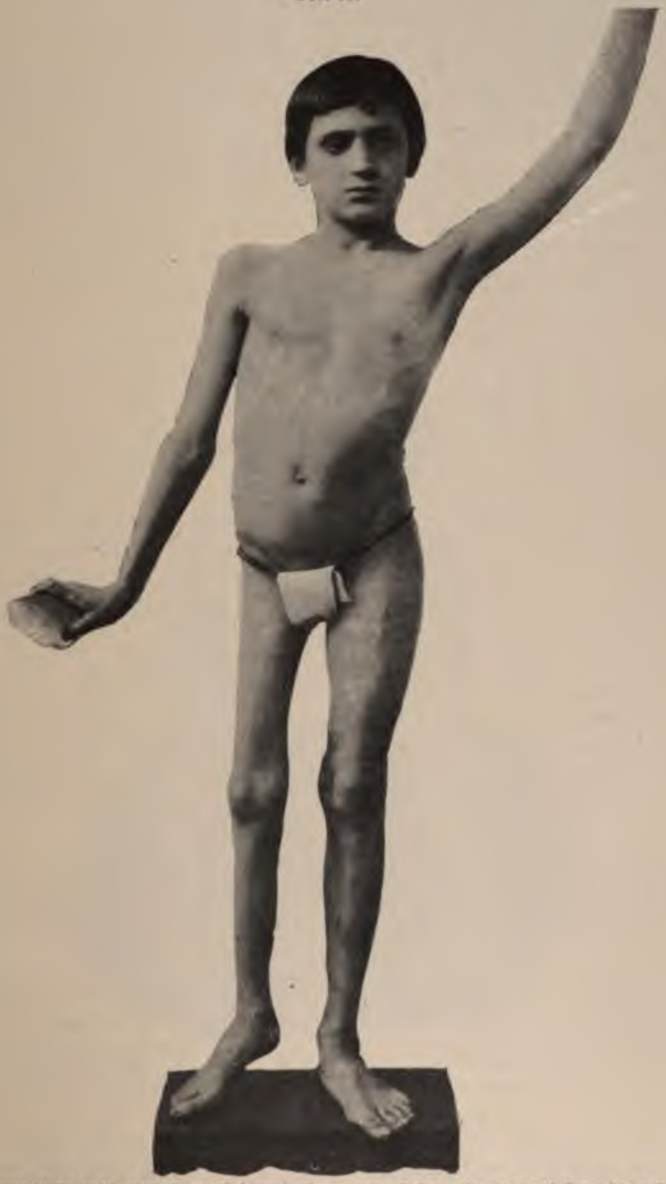
FIG. 56.



Extreme atrophy of the shoulder, arm, and forearm in an adult who suffered from an attack of infantile paralysis at the age of three.

The circulation in the affected limb is considerably impaired, and it is cold, blue, and flabby, but not œdematous. In some cases the bone is subsequently hampered in its growth, so that the limb is shorter and more slender than its fellow in after life. (See Fig. 56.) While the description just given of an acute onset with fever

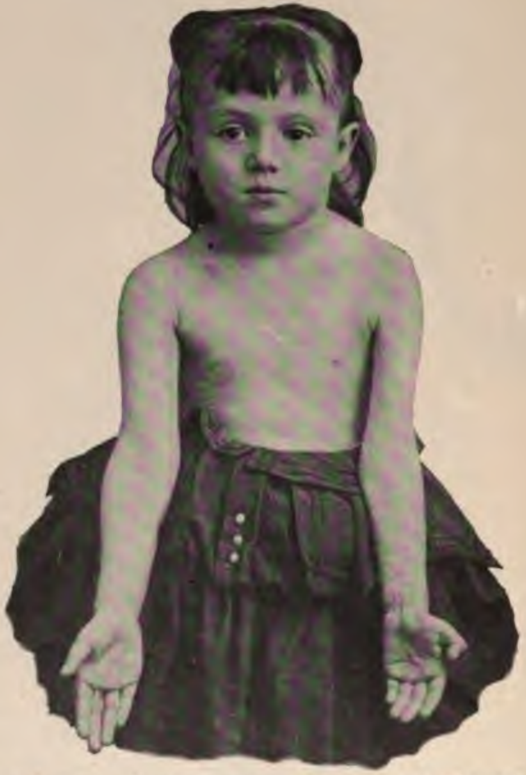
FIG. 57.



Infantile paralysis, with atrophy and impaired growth of the right leg, and drop-foot; four years after the onset.

applies to about three-quarters of the cases of anterior poliomyelitis, there remains one-quarter in which there is no febrile onset. Of 166 cases in my clinic, 120 began with fever and 46 began without fever. Sinkler reported 178 with fever, 40 without fever. In these cases the child while in a state of perfect health is suddenly paralyzed in one or more limbs. It gives no sign of pain, it does not appear to be ill, and the paralysis surprises the mother by its sudden onset. In these cases the paralysis is soon followed by atrophy and by vasomotor paralysis. It is not attended by pain or tenderness on motion, and usually decreases to some extent, leaving the limb, however, in part permanently paralyzed.

FIG. 58.



Infantile paralysis and atrophy of the left arm two years after onset. The partial luxation of the humerus is evident; and also the *main en griffe.*

These two types of onset of the disease are evidently quite distinct from one another, and their pathological basis is probably different, as has been already stated.

After the onset is over there is a slow progressive improvement up to a certain point, and then the permanent condition of paralysis is found to vary greatly in different cases.

The location of the paralysis is usually in the legs, and here two types of the disease may be recognized—the leg type and the thigh type. In the leg type the peronei alone or with the anterior tibial muscles are commonly affected, although the posterior tibial group may share in the paralysis or may even be as fully paralyzed as the others. As the paralysis remains, deformities of the ankle and foot will appear, the form of talipes developed depending upon the muscles chiefly paralyzed. In the thigh type, the psoas and iliacus muscles and the glutei and muscles about the thigh are those chiefly affected, the muscles on the inner side of the thigh and the muscles below the knee often escaping. In these cases the leg hangs like a flail from the body, and cannot support the weight at all. In some cases nearly all of the muscles of the lower extremity are paralyzed, and the atrophy is uniform throughout the limb. In these severe cases it is not uncommon for the muscles of the back and abdomen to share in the paralysis and atrophy.

When the arms are attacked two types of paralysis have been described, the upper-arm type and the lower-arm type. In the upper-arm type the muscles about the scapula and the deltoid, the biceps, and supinator longus are paralyzed and atrophic, and consequently the motions of the shoulder-joint and elbow-joint are seriously hampered. In these cases the shoulder-joint is unduly movable and the head of the humerus falls out of the socket. In the lower-arm type the muscles below the elbow are invaded, the flexors or extensors of the wrist and fingers, or both together, are affected, the supinator longus escaping. In other cases the interossei and the thenar and hypothenar muscles of the hand are paralyzed, while the long flexors and extensors escape. Occasionally a combination of upper-arm and lower-arm types occurs, in which case the entire extremity is useless. The upper part of the trunk is occasionally involved in the paralysis together with the arms. The muscles of the back and trunk are rarely the only ones permanently paralyzed.

In a very few cases the entire muscular system of the body appears to be affected by this disease; both legs, the trunk, and both arms are more or less paralyzed; but even in these cases a careful examination will show that the degree of the paralysis and atrophy is not the same in all the muscles. The relative frequency of paralysis in different parts of the body is shown in the following table (Table VI.). The paralysis is rarely, if ever, exactly symmetrical when both legs or both arms are involved.

TABLE VI.—*The Distribution of Permanent Paralysis.*

	Duchenne.[1]	*Seeligmüller.*[2]	*Sinkler.*[3]	*Starr.*	*Total.*
Both legs	9	14	107	54	184
Right leg	25	15	63	31	134
Left leg	7	27	62	37	133
Right arm	5	9	5	11	30
Left arm	5	4	8	6	23
Both arms	2	1	1	5	9
All extremities	5	2	35	9	51
Arm and leg same side	11	2	26	9	48
Arm and leg opposite sides	2	1	1	6	10
Trunk	1	...	22	4	27
Three extremities	...	...	10	5	15

In addition to the paralysis and atrophy, there is a loss of reflex action at the level of the lesion in every case. The skin reflexes usually return after a time, but the tendon reflexes are absent for a long period, even when a partial recovery of the muscle involved has taken place. Thus the knee-jerk is uniformly absent when the thigh muscles are paralyzed, and the elbow-jerk and wrist-jerk when the arms are affected.

Sensation is preserved in almost every case, but I have so frequently observed a permanent hypersensitive condition to painful impressions in the paralyzed limb that I cannot but believe that the lesion in the gray matter affects the pain-sense tracts in their passage through the cord at the level of their entrance, and has a relation to this symptom. There is marked vasomotor paralysis and lack of vasomotor response in the limb to applications of heat and cold.

Deformities of the joints are a common sequel in infantile spinal paralysis. The approximation of articular surfaces is secured, in part, by the normal tension of the muscles, especially at the shoulder, hip, and knee, and hence paralysis of the muscles controlling these joints is attended by relaxation and a greater degree of mobility than is normal. Thus the head of the humerus falls from its socket when the deltoid is paralyzed, and abnormal extension of the knee is often seen in the upper-leg type of paralysis. After some months of paralysis the muscles which are the natural opponents of the paralyzed muscles are apt to become permanently contractured, and this also occasions deformities. The action of gravitation on a flaccid part of the limb combines with the contracture in the case of the foot to increase the deformity there, and hence all forms of talipes may ensue upon infantile paralysis. Deformities of the wrist are also observed, but these are not common. Curvature of the spine from paralysis of the muscles of the back is frequently seen, all varieties having been described. Its most common cause is the shortness of one leg, due to arrest of its growth. Such curvatures differ from those due to bone disease in the fact that they do not persist during suspension of the body by the head and arms. It is one of the most important points in treatment to prevent the development of these deformities.

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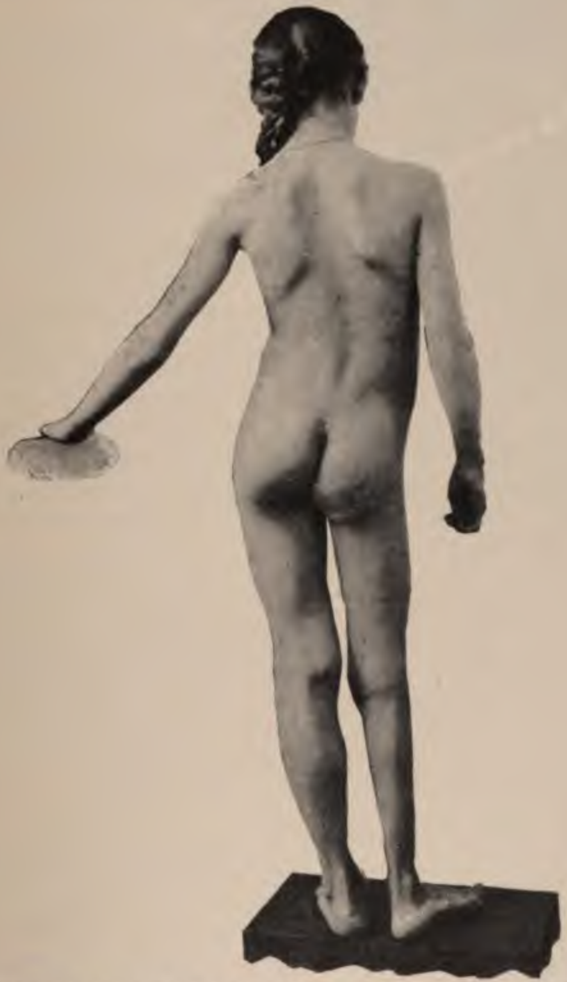
[1] Archives gén. de Méd., 1864, p. 38.

[2] Gerhardt's Handbuch d. Kinderkrankheiten, 1880, vol. v. p. 1.

[3] Keating's Cyclop. of Children's Diseases, 1890, vol. iv. p. 695.

The progress of the disease in any case may be divided into stages. After an acute onset there is a stage of maximum intensity lasting from one to six weeks, and followed by a period of steady improvement which may extend from six months to a year. Then follows the permanent chronic condition, in which the normal growth of the child may lead to a slow development of the limb, but not to any change in its power of use. It is very rare for a complete recovery

FIG. 59.



Infantile paralysis with atrophy of the right leg. The curvature of the spine is secondary to the shortening of the leg.

to take place after an attack of infantile paralysis. Even in the lightest cases there is usually some weakness, slight atrophy, and coldness left, and one or two muscles will be particularly feeble. In the majority of cases considerable permanent paralysis remains, requiring the use of apparatus to assist the action of the limb and to prevent deformities. Death has occasionally occurred during the acute onset, but is very rare, and once this stage is passed there is nothing in the disease to threaten life.

It is the chief characteristic of the atrophic paralysis in this disease that it selects certain muscles to the exclusion of others. This selection bears no relation to the arrangement of muscles in the limb or to the conjoint action of muscles in producing any definite movement. It is wholly dependent upon the arrangement of the groups of cells controlling the muscles in the anterior horns of the spinal cord. The exact localization of the disease can be determined by referring the symptoms in any case to Table I. in the chapter on the Diagnosis of Spinal-cord Diseases, page 173.

Diagnosis. There is no difficulty in recognizing the disease, and it is hardly likely to be mistaken for anything else. Occasionally a child will be attacked with acute articular rheumatism, and, on account of the pain in the joints, will be unwilling to move the limbs, and thus may be thought to be paralyzed. A careful examination should soon demonstrate the real condition, for acute rheumatism never causes any atrophy or paralysis, and the local tenderness in the joints, the sweating, and the lack of coldness of the limbs may also aid in the diagnosis.

Rachitis, sometimes caused in infants living in healthful and comfortable surroundings by the use of artificial patent foods containing considerable sugar, may lead to a sudden febrile onset, with much pain and tenderness in the limbs and unwillingness to move. But the child is not really paralyzed, and the tenderness of its bones, the appearance of the gums and the sweating, as well as the lack of limitation of the pain and immobility to one or two limbs, should prevent this disease from being mistaken for infantile paralysis.

In some cases of anterior poliomyelitis there is considerable pain felt in the limbs, and some tenderness of the surface and of the muscles. The existence of pain during the first two days of the disease occasionally leads to mistakes in diagnosis. Thus Marsh[1] records a case of a child aged five years, who was suddenly attacked with pain in the left leg extending down the thigh to the knee. The limb was flexed, abducted, and rotated outward, and any motion was painful; hence the case was recorded as acute hip disease, but closer examination showed the hip-joint to be quite freely movable, and after two days, when the pain had passed away, the case was found to be one of infantile paralysis. The fever and general constitutional disturbances present at the onset had rendered the diagnosis obscure.

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[1] Lancet, January 16, 1897.

In painful cases it has been suggested that a neuritis may accompany the poliomyelitis. The existence of pain alone is not sufficient to warrant this conclusion, inasmuch as we now know that in the early stages there is a congestion of the gray matter of the cord which is sufficient to explain the pain. If, however, the pain continues and tenderness develops in the muscles and nerves, it is probable that a neuritis has developed due to the same infectious agent which has caused the poliomyelitis. It is to be remembered that polyneuritis is usually a disease affecting the extremities symmetrically and causing drop-wrist and drop-foot; that the distal parts of the extremities are more severely paralyzed than the proximal parts; that there is no such selection of muscles paralyzed as in poliomyelitis, and that there are usually sensory disturbances of a permanent nature, anæsthesia and analgesia, or ataxia, in addition to the pain and tenderness along the nerves; hence in the acute stage of onset a polyneuritis should not be confounded with a poliomyelitis. When polyneuritis accompanies poliomyelitis the clinical picture will be made up of a combination of the symptoms of both affections (see page 110).

A localized injury of the brachial plexus (Erb's paralysis), causing paralysis of the deltoid, biceps, coracobrachialis and supinator longus is not uncommon in infants, and might be mistaken for infantile palsy. The history of trauma during delivery and the local anæsthesia in the distribution of the circumflex nerve should, however, correct the mistake (see page 61).

Prognosis. The prognosis in anterior poliomyelitis is always grave. Patients do not often die of the affection, but they rarely escape a permanent paralysis in some part of the body. It is true that in the majority of cases the original paralysis subsides, so that there is an apparent improvement of a considerable degree. Thus a patient who has originally been paralyzed in both legs may recover the power in one leg entirely, and may be left with a condition of paralysis in the peronei or in the anterior tibial group of the other leg, so that the terminal condition is very much less severe than that at the onset. As a rule, the limb that is affected never entirely regains its power, and usually shows some atrophy and shortening; for the growth of the limbs is hampered by the existence of the disease, and hence in a growing child the unaffected limb outgrows the other. It is thought that an electrical examination may afford some ground for a prognosis. It is believed that the muscles which respond to the faradic current three weeks after the onset of the disease will eventually recover, while those that fail to respond to this current at that time will always be somewhat impaired in power. The loss of faradic reaction, however, is not an indication that these muscles will be totally paralyzed, since the faradic reaction has been known to return in a muscle a year after it has been lost, yet such a muscle never recovers completely its size or power. The prognosis is much better in the cases which begin with fever than in those which do not.

Treatment. The treatment of infantile spinal paralysis in the acute stage consists in keeping the child quiet in bed and applying a mild form of counter-irritation along the spine, which is best done by a paste of mustard 1 part and flour 3 parts, applied in a poultice along the back and removed as soon as the skin is reddened, and then renewed after three hours, so that for at least a week there shall be continual counter-irritation without the discomfort of a blister. The frequent application of dry cups along the spine may be used to produce the same effect. Repeated sponging with alcohol and cool water is indicated in the cases in which the temperature is above 101° F., but phenacetin or antipyrine is not to be used unless the temperature reaches 103° F. There is some advantage to be gained from the internal use of ergot. The dose of ergot is 10 minims of the fluid extract for a child below the age of two years, every four hours, and 2 minims more for each additional year. Iodide of potassium may be given in 1-grain dose in the early stage, and moderate doses of salicylate of sodium (2. gr.) or of quinine ($\frac{1}{2}$ gr.) may be used for a child of two years. If the child is in much pain or has convulsions, bromide of sodium (5 gr.), with or without codeine ($\frac{1}{10}$ gr.), may be employed as a symptomatic remedy. The general treatment of febrile conditions, a light diet and laxatives, is not to be neglected. The best laxative is castor oil ℥j, glycerin ℥j, cinnamon-water ♏x, given with an equal amount of lemon juice sweetened. Rest in a prone position in bed is better than constant lying upon the back.

When the acute stage is passed there is little to be done during the second week excepting to nourish the child well and to keep the paralyzed limb warm. Iodide of potassium in 1-grain to 3-grain doses may be administered three times a day.

When the paralysis begins to subside spontaneously it is well to administer strychnine in full dose, $\frac{1}{50}$ gr., three times a day for a child of three years of age. This remedy is best given at intervals, and not continuously, and it is my rule to use it for one week, and then to intermit for three days. The condition of mechanical irritability in unparalyzed muscles, as determined by percussion with a hammer, is a good indication of the degree of effect being produced by the strychnine, and the drug may be increased until it causes a distinct increase in this irritability. It is to be remembered, however, that twitching of the limbs or stiffness of the back, usually indicative of an effect of strychnine, is not to be relied upon in infantile paralysis when the muscles are paralyzed. Whether general tonics, such as cod-liver oil, hypophosphites, or arsenic, have any effect of a favorable kind may be left to the judgment of the physician in each individual case.

The most important indication during the stage of regression is to preserve the nutrition and function of the paralyzed muscles, and this is to be attained by skilful massage, by hydrotherapy, or by the use of electricity. Massage is of the utmost importance in these cases, and should be given once or twice a day with care, combined with such attempts at active movement as the child is able to make. Among the poorer classes it is well to instruct the mother how to give this, so that it may be given with persistence. The massage should not be of the hardest kind, and yet should be sufficient to stimulate the circulation in the limbs and to promote the lymphatic and venous flow. Next to massage mechanical devices which induce the child to make use of the weakened limb are to be employed. A household gymnasium adapted to each individual case can easily be devised by the physician, and if such exercises are made of the nature of play to the child's imagination, much good will be derived from its own efforts.

Hydrotherapy is also an important aid in treatment. The general circulation in the cold and flabby limb may be aided by warm baths, and it is my rule to have these children play in warm water, temperature 99° F., for half an hour twice daily. This warm bath may be followed by a cooler sponging and brisk rubbing, but cold water should not be employed in the bathing of these children, as the temperature of the paralyzed limb is always below that of health, and the vasomotor paralysis prevents the quick reaction which is so beneficial in other conditions. Proper protection of these limbs by extra flannel clothing is always advisable.

Electricity is a valuable agent in the treatment of infantile paralysis, but a clear statement of its use should be made by the physician to the family. Electricity has no influence whatever upon the course of the disease. It does not affect the lesion in the spinal cord, either to decrease the hyperæmia or to increase the nutrition of the nerve centres. Applications, therefore, of galvanism to the spine are absolutely useless. But applications to the muscles may be of distinct service in two different ways—first, by causing their contraction, and thus exercising them when voluntary exercise is impossible, and, secondly, by promoting the chemical changes in the muscle that are essential to growth and nutrition.

Examination in any case will show a certain number of muscles in the paralyzed limb that respond to faradism. These muscles will eventually recover entirely, yet the tone of the muscle and its strength can be kept up during the period of improvement by means of exercise with either the faradic or galvanic current. It is quite well proven that just as exercise of a healthy arm will markedly increase the size of the biceps muscle, so applications of faradism regularly to a muscle that it will contract will increase the size of this muscle; hence to the weakened muscles which still respond to faradism an application of the faradic current for about ten minutes once or twice a day will be of service. The majority of the paralyzed muscles do not respond, however, to faradism, and it is time wasted to apply the faradic current to these muscles. They do respond, as a rule, to galvanic interrupted currents, the positive pole being placed over the muscle and the negative upon the limb at a short distance above. The interruptions should be made by an electrode held in the hand and provided with a finger-key, and each muscle should be treated for about three minutes daily, fifty to sixty interruptions being made per minute by the finger. The strength used should be the least which will secure contraction in the muscle. When interruptions of the current do not produce a prompt response alternation of the current may be employed by reversing the current rapidly by means of the pole-changer on the battery. It is to be remembered that in this disease the application of electricity is more painful than in health. It is also to be remembered in applying electricity to children that their confidence must be gained, and that, if they are frightened at the first application, subsequent treatment will result in a continual struggle. It is my custom, therefore, to begin a course of electrical treatment to a child by several applications of the sponges and electrodes while no current is passing, thus accustoming the child to the apparatus and gaining its confidence. After two or three such applications it will be possible to use a weak current, and then day by day to increase its strength until by the end of ten days the necessary strength is being used. In this way a daily struggle, with the result of unsatisfactory and probably useless applications, can be avoided; and the parents' consent obtained to a course of treatment which they would eventually object to if every application resulted in a struggle. Any intelligent mother or nurse can be taught to give the galvanism or faradism to a child in this manner, and it is best to interest the attendant in the treatment from the beginning, and to instruct her carefully, so that within a week the treatment can be left entirely in her hands. Such an application of electricity is to be made daily or twice a day for two or three years. Spontaneous recovery will have been reached at the end of the first year, but even after this time these muscles may be brought into a condition of hypertrophy by means of continued exercise. When, however, a child is quite able to move voluntarily with some force any paralyzed muscle, it is far better to rely upon voluntary exercises than upon electrical applications. If no effect is obtained from massage, bathing, and electricity in a muscle at the end of a year there is no use in continuing the treatment of that muscle, as it will never recover, its nerve cells being entirely destroyed.

The use of braces plays a great part in the treatment of infantile paralysis in the chronic stage. It is to be remembered that many weak muscles can do their work only when the limb is placed in an advantageous position or when they are assisted in their action. Many of the muscles have, as part of their function, to keep the joints in place, and this part can be supplied by properly adjusted braces; hence an apparatus may enable the child to use a muscle or to move a joint that it could not do if the joint were unsupported. Again, the result of paralysis of one group of muscles is to allow the joint to be bent by its opponent or to yield to the influence of gravitation, and hence the paralysis is often followed by deformity if a brace is not applied early to correct this tendency. There is no disease in which orthopedic apparatus is of more service than in infantile paralysis, and it cannot be applied too early, as it may prevent the development of contractures and of deformities. There is no stage in which it is too late to fit a brace, for even if these deformities have occurred tenotomy may be employed to straighten and adjust a joint, and then the limb can be fixed by the brace in a proper position. But every case has to be treated skilfully in accordance with its own condition, and the ready-made braces of the shops are often worse than none. Hence for each case a special apparatus must be fitted under the direction of an orthopedic surgeon, and it is to be remembered that in a growing child such apparatus must be constantly readjusted, its length and size being changed from month to month in accordance with the development of the limb.

In many cases of deformity where there is a strong contracture of a fairly healthy muscle overcoming the weak paralyzed muscle the question of tenotomy will arise. Such tenotomy will of course result in a temporary replacement of the deformed joint to its natural position, but unless the joint can be held by a brace in this position, tenotomy alone will be of no permanent service. Hence tenotomy is only to be regarded as a preliminary in some cases to the proper application of apparatus. Apparatus has also been devised (especially in the treatment of infantile paralysis of the hands) by means of which weakened muscles may be reinforced by elastic bands so applied as to take the place of the paralyzed muscle. Thus a dropped-wrist or a paralysis of the extensors of one side of the wrist can be somewhat relieved by a series of elastic bands attached to finger-tips or to rings and to the elbow and running through a bracelet at the wrist. Dropped-foot may also be similarly remedied. Such devices, however, are usually discarded after a time, as they are more cumbersome than useful. Apparatus is especially applicable to spinal curvature of the paralytic type, and in any case in which the body or back muscles are involved at the onset it is well for the child to wear a corset in order to prevent the development of some form of curvature. A thick cork sole will prevent the curvature due to a short leg.

It has been proposed to divide longitudinally tendons of certain healthy muscles and attach one-half to the severed tendons of paralyzed muscles about the knee, ankle, wrist, and elbow, in order that the healthy muscle may be made to do the work of the muscle which is paralyzed, and many successful attempts in this direction have been reported. I have seen in a case of paralysis of the peronei, permanent benefit in a remarkable degree by this method of treatment, in which a part of the posterior tibial tendon was attached to the cut peroneus longus tendon. I have also seen paralysis of the anterior tibial muscle relieved by attaching its tendon to the tendon of the long peroneal muscle. I have seen paralysis of the soleus improved by attaching its tendon to the deep muscles beneath it, and I have seen several cases of great improvement in movements of the fingers and wrist by attaching the tendons of paralyzed muscles to those which were healthy. This is a method of relief which is rational and deserves wide adoption.[1]

CHRONIC ANTERIOR POLIOMYELITIS.

Chronic Atrophic Paralysis. Progressive Muscular Atrophy.

History. A condition of progressive paralysis with atrophy was first described by Sir Charles Bell in 1836; it was not referred to a lesion of the spinal cord, however, until 1850, when Aran[2] studied it carefully. His statements were added to by Duchenne in 1853. But the form of progressive atrophic paralysis, termed progressive muscular atrophy of Aran-Duchenne, was soon found to be but one of several types of the disease. Duchenne in 1853[3] described another type, which he named *paralysie générale antérieure subäigue ascendante*, a disease beginning with paralysis and atrophy in the legs, and gradually invading the trunk and the arms, and finally causing death from respiratory paralysis. For many years all cases of chronic atrophic paralysis were referred to these two classes, and were supposed to have as a basis an affection of the anterior gray matter of the cord.

But between 1860 and 1870 numerous cases of atrophic paralysis were reported in which no spinal lesion was to be found. And gradually it became evident that some forms of this disease were dependent entirely upon changes in the muscles. To Friederich[4] and the German school must be given the credit of separating the muscular dystrophies from the forms of spinal paralysis.

In 1872 Charcot[5] and the French school discovered amyotrophic lateral sclerosis and showed the differences between it and progressive muscular atrophy. And finally in 1882 Dejerine and others brought forward many facts to prove that numerous cases formerly supposed to be due to spinal lesions were really due to multiple neuritis (see page 117).

A chronic atrophic paralysis characterized by a slowly progressing weakness, and atrophy beginning in one part of the body and advancing to another part, may occur under the following conditions:

First: Chronic anterior poliomyelitis.

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[1] Tubby. Lancet, March 28, 1903.

[2] Arch. gén. de Méd., xxiv. 42.

[3] Traité de l'Electrotherapie localisée.

[4] Ueber progressive Muskelatrophie. Berlin, 1873.

[5] Leçons sur les maladies du système nerveux. Paris, 1880, ii. 192.

Second: Amyotrophic lateral sclerosis, (*a*) beginning in the spinal cord and ascending to the medulla and pons; (*b*) beginning in the medulla and pons as bulbar paralysis, and descending to the spinal cord.

Third: Multiple neuritis.

Fourth: Muscular dystrophy.

In the present chapter attention will be confined to chronic anterior poliomyelitis, other conditions causing atrophic paralysis being considered elsewhere.

Pathology. The pathological changes present in this disease consist of a slowly advancing atrophy in the primary motor neurones of the cord, cell bodies, dendrites, and axones degenerating together. These cells, as already stated (p. 171), lie in groups in the anterior horns and in the central gray matter, and the lesion affects these groups in different degrees.

We have several clinical types of chronic anterior poliomyelitis, due to the fact that the lesion may begin in different parts of the spinal cord.

In the first type the atrophy begins in the lower groups of cells of the lumbosacral region, and extends to all the groups in the lumbar enlargement. The paralysis begins in the peronei and anterior tibial groups of muscles, then advances to the adductors of the thigh and glutei, and finally invades all the muscles of the legs, but does not extend to the arms.

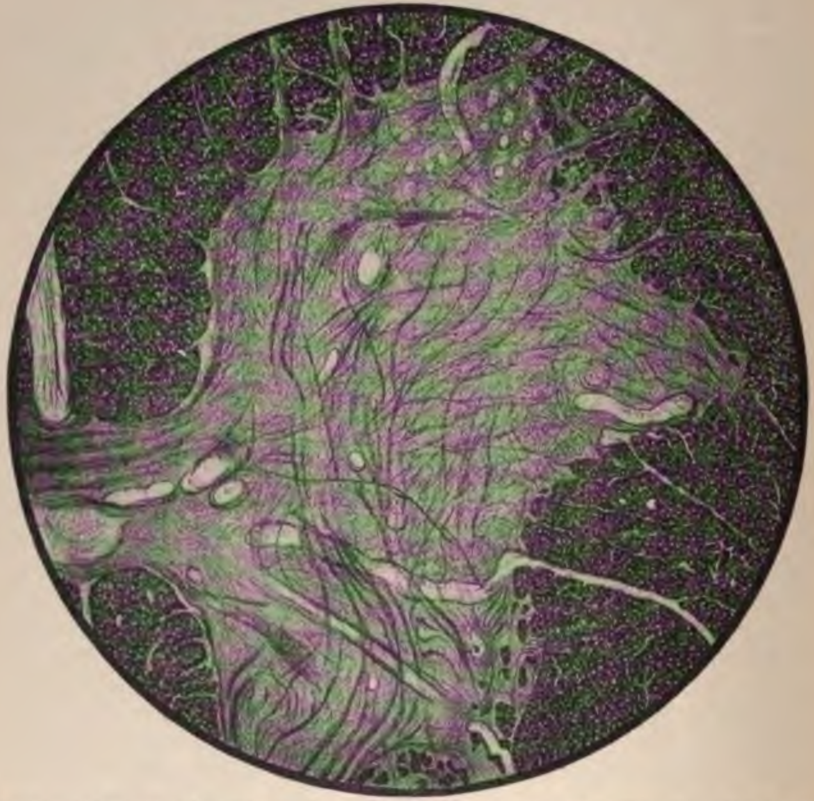
In the second type of the disease (Duchenne's subacute ascending paralysis) there is a progressive atrophy of the cells, beginning in the lumbosacral region and advancing gradually up the cord until all the motor cells are involved. The cells in the sacral region, in which the lesion begins, show a more advanced and complete atrophy than those in the cervical region, though at the end of the disease, in case the patient is not carried off by some intercurrent affection, a large majority of the cells of the anterior horns have disappeared.

In the third type (the progressive muscular atrophy of spinal origin of Aran-Duchenne) the atrophic process is limited to certain groups of cells in the cervical region of the cord, especially in the first dorsal, eighth and seventh cervical segments which govern the hands. After it has progressed to a considerable extent in these segments, the groups of cells in the fifth segment, which control the deltoid muscle, and the adjacent groups controlling the biceps, coracobrachialis, and supinator longus muscles, become affected, and then finally all the groups of the cervical enlargement are attacked by the disease. Later the same lesions appear in the lumbar region in the various groups of cells, and the paralysis extends to the legs.

The lesion in all these types is similar. The difference in the clinical symptoms is wholly due to the difference of location, different groups of cells being first attacked or progressively invaded in the three types of case. The difference between the subacute and chronic cases is only the difference of rapidity in progress, and no hard-and-fast line can be drawn between the two.

The exact lesions in these affections consist in changes which are only visible to the microscope. There is no deformity or atrophy of the spinal cord on inspection, and there is no change in the membranes of the cord. The anterior nerve roots may appear to be slightly atrophied, and are liable to tear more easily in the process of extraction of the cord. On microscopic examination there is not to be seen any congestion of the cord or any exudation of cells within the substance, but a simple progressive atrophy in various stages in the cells of the anterior horns and of the central gray matter. There is no breaking of the dendrites, no swelling of the axones, no intracellular vacuolization, no hernia of the nucleus, and no diffuse chromatolysis. There is no trace of capillary hemorrhages.

FIG. 60.



The anterior horn in the lumbar region in a case of chronic anterior poliomyelitis. Total disappearance of the cells, and thinning of the network of fibres within the horn. (Oppenheim.)

The cell remains with all its characteristics, but is simply shrunken. In the early stage the body of the cell is small and its nucleus is diminished in volume, but the cell retains its polygonal form, merely becoming too small for its lymphatic space. Its chromatophile granules appear small, almost like a mass of dust within the cell, and may be thicker about the nucleus or in the periphery. Collections of pigment in the cell appear in large quantity. Little by little the cell diminishes until the only thing left is a round cellular nucleus with a small nucleolus or a mass of pigment granules. (See Plate III., F. G.) The lesion attacks not only the large and small cells which give origin to the anterior nerve-root fibres, but also the large and small cells which give rise to the association fibres passing into the antero-lateral column of the spinal cord. The cells of the column of Clarke are not affected.

As a consequence of this atrophy of the cells there is a corresponding atrophy and disappearance of their dendrites and axones. As the dendrites disappear the fine plexus of fibres in the gray matter becomes less noticeable. As the axones atrophy there is a shrinkage in the antero-lateral tracts of the cord due to a disappearance of the anterior motor-root fibres, and also of the association fibres that pass to other levels of the cord. The atrophy of the fibres within this column is diffuse, fibres here and there through the entire antero-lateral column being found atrophied; hence, although no lesion is apparent by the Weigert stain, the Marchi method reveals this diffuse degeneration. A picrocarmine stain demonstrates very slight sclerosis of the antero-lateral column adjacent to the anterior horn of the cord on all its sides. The other tracts are normal. There is marked degeneration and atrophy in the anterior nerve-root fibres and in the nerves to their termination in the muscles. There is a simple atrophy of the muscular fibres in the muscles which are paralyzed. This atrophy consists of a granular disintegration or a disintegration of the muscles, with fine fatty deposits and disappearance of striation and a simple atrophy of the muscular fibres. Occasionally a single hypertrophied muscular fibre may be found.[1]

Symptoms. The symptoms of chronic anterior poliomyelitis differ in the different types of cases. First, in chronic ascending paralysis we have a slowly advancing paralysis beginning in the peroneii muscles, first in one leg, but within two or three months of the onset appearing also in the other leg and causing a sensation of weakness in walking, with a tendency to dropping of the feet. The paralysis corresponds exactly with the degree of atrophy present. After some months the weakness appears in the anterior tibial group of muscles, and then a fully developed drop-foot appears. The patient walks with difficulty and steps high in order to avoid stumbling over his toes. Any dorsal flexion of the foot is impossible when the patient is lying down, and while standing he cannot lift the toes from the floor. The paralysis and atrophy go on little by little, measurements showing a reduction of perhaps one-quarter of an inch every month in the calf. The mechanical excitability of the muscles is diminished, but not lost, and there is very often a fine fibrillary twitching in the muscles, causing a wave-like movement of the skin, especially when the muscles are exposed to cold or are percussed. Patients may perceive fatigue on slight exertion and may have slight muscular pains in the leg, but they have no sharp pains and absolutely no sensory disturbances. After several months the paralysis advances to the adductors of the thigh and to the glutei muscles. Then walking becomes even more difficult, going up stairs becomes impossible, and it is with difficulty that the patient rises from the chair. In one case still under my observation two years elapsed before this stage was reached. Then about simultaneously the posterior tibial muscles of the leg and the anterior muscles of the thigh are invaded, and also the psoas and iliacus muscles, and when the paralysis in these muscles is advanced to a moderate degree the patients are no longer able to walk. The measurements of the legs are by this time reduced several inches, and not uncommonly the atrophy becomes extreme. The legs feel rather cold to the touch, but there is rarely any œdema, and there is no tendency to bed-sores. The disease may not go beyond this point, and for years the patient may live in a state of paraplegia. There is no tendency to any affection of the bladder or rectum, and the muscles of the back may escape.

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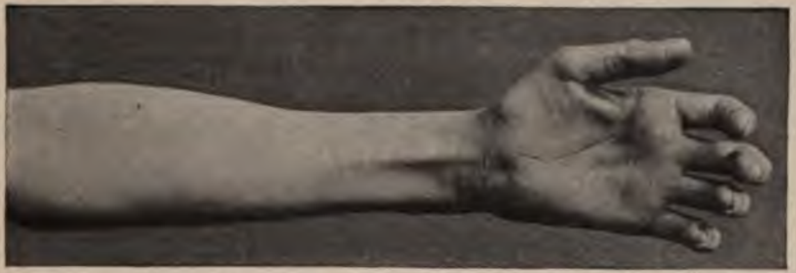
[1] Recent cases with careful pathological observations in this disease have been reported by Phillipe and Ceston before the Neurological Section of the International Medical Congress, Paris, 1900; also by Raymond and Rickling at the same Congress; and by J. B. Charcot, Thèse de Paris, 1892.

In the second type—Duchenne's ascending paralysis—there is a tendency to progression upward of the affection. The muscles of the back and trunk become involved, the patient can no longer sit upright in bed or on a chair, and usually the muscles of the shoulders and of the hands become invaded simultaneously, and inability to use the upper extremities gradually develops. The course of the atrophy of these cases may be quite similar to progressive muscular atrophy of the Aran-Duchenne type, and finally bulbar paralysis may ensue. These patients commonly die of some intercurrent disease, pneumonia being the most common cause of death, especially if the respiratory muscles become affected.

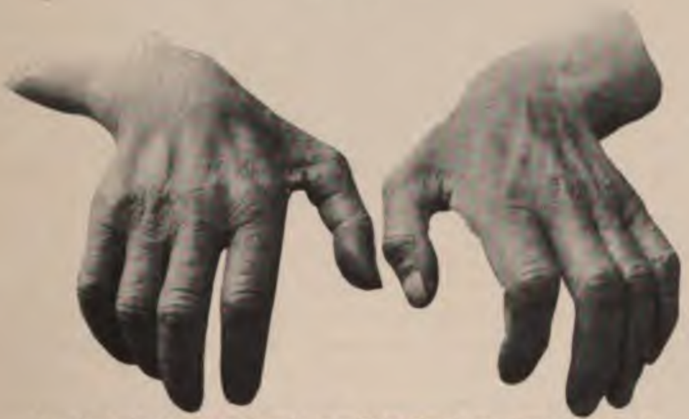
The third type of chronic anterior poliomyelitis is the type described by Aran and Duchenne. The patient first notices an inability to move the muscles of the thumb of one hand, adduction being imperfect. Almost simultaneously there is a paralysis of the abductor indicis, and consequently the patient cannot separate the forefinger from the middle finger. The paralysis of these small muscles is noticed in the finer movements of writing, playing the piano, buttoning the clothes, or picking up small objects, and this disability is the first thing which attracts the patient's attention. As the disease goes on the paralysis extends to the other fine muscles of the hand, the interossei, lumbricales, and the muscles moving the little finger. And parallel with the paralysis there is an atrophy in all these muscles, causing a flattening of the thenar and hypothenar eminences and a distinct thinning of the hand, producing a sulcus upon its outer side. The thumb can no longer be touched to the tip of the fingers, and, as the interossei are flexors of the first phalanx, flexion of the fingers is confined to the second and third phalanges; the hand, therefore, cannot be closed firmly. A characteristic abnormal position is soon assumed by the hand as a result of this paralysis of the interossei. They no longer afford any opposition to the long flexor and extensor muscles. The long flexors flex the second and third phalanges, the long extensors extend the first phalanx, hence the hand is thrown into a position called *main en griffe* or claw-hand. (See Fig. 62.) And as the interosseii are much atrophied the tendons of the long muscles stand out upon the hand, forming distinct cords which may be felt and seen. At the same time a paralysis of the thenar muscles results in a hyperextension of the first phalanx of the thumb,

FIG. 61.



Hand and forearm in chronic spinal muscular atrophy, showing especially wasting of the thenar and hypothenar eminences and of the abductor indicis. (Dercum.)

FIG. 62.



Atrophy and paralysis with *main en griffe* in chronic anterior poliomyelitis.

with some rotation outward, so that the ball of the thumb is in a plane parallel with that of the palm.

As a rule, one hand is invaded some months before the other, and in several cases I have seen the disease come to a standstill when only one hand was affected; but, as a rule, there is a tendency to a gradual progress of the paralysis and atrophy, until both hands are almost useless. In one case three years elapsed before this condition was reached. But before this stage is reached a progressive atrophy appears in the deltoids, first in one, and then in the other shoulder. In consequence of this paralysis abduction of the arms above the horizontal line is impossible, and when the patient is stripped it is evident that almost all abduction of the arm is produced by rotation of the shoulder-blade by the serratus magnus muscle.

The paralysis then extends from the deltoid to the biceps, brachialis anticus, and supinator longus muscles. The supraspinatus and infraspinatus are also affected, producing a marked atrophy about the shoulder-blade, and later the teres and subscapularis become affected. In this condition the patient is completely paralyzed in the arms, which hang like flails at the side, and are of very little use, flexion at the elbow being impossible by voluntary effort.

As the disease progresses beyond this point the atrophy attacks the muscles of the body about the shoulders. The serratus magnus is paralyzed, and then the shoulder-blade sticks out from the side of the body like a wing. The rhomboids, the levator anguli scapulæ, and the lower half of the trapezius become paralyzed, the shoulder-blade cannot be moved, and the muscles of the back become so atrophied that all the bones are visible. Even in the extreme cases, however, certain muscles escape for some unknown reason—namely, the upper part of the trapezius between the occipital bone and the clavicle, the triceps, latissimus dorsi, and the lower half of the pectoralis major. As these muscles stand out in contrast to the atrophied muscles about them and contract because of the lack of opposition, deformity of the shoulders and of the neck is quite evident.

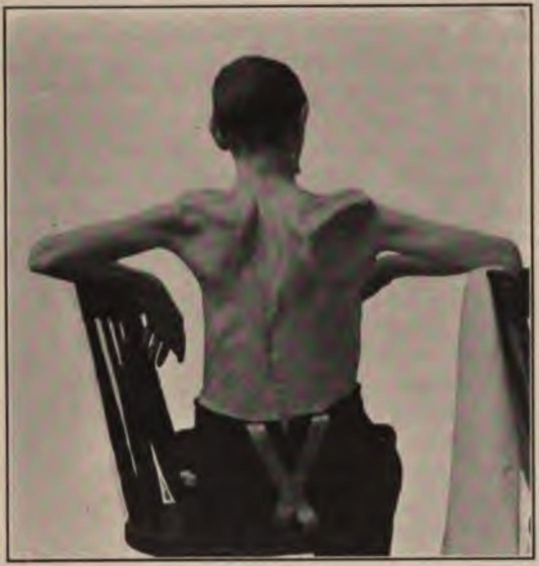
As the disease extends the muscles of the neck are next invaded, and, as these hold the head in an erect posture, gravitation leads to a falling forward of the head, so that the chin rests upon the chest. This falling forward of the head is usually intensified by the contraction of the sterno-cleido-mastoid muscles, which are not affected by the disease.

In the last stage of the case the paralysis and atrophy extend to the intercostal muscles and to the muscles of respiration, so that all respiration becomes diaphragmatic. As a rule, patients die from some intercurrent disease when the atrophy and paralysis have reached this stage; but in case they live the paralysis may extend to the legs, causing a condition of paraplegia of progressive type such as has been already described in the first class of cases. In a number of cases of both types there has been an extension of the paralysis and atrophy to the muscles of the face, of the tongue, and of the throat, producing a gradual difficulty in articulation, in swallowing, and in all movements of the face, and death by suffocation or choking.

The symptoms of this complication are more fully described under the head of bulbar palsy.

In all these forms of paralysis with atrophy there are frequent fibrillary contractions in the paralyzed muscle, which are both spontaneous, and can be caused by percussion or by exposure of the limb to cold. There are also progressive electrical changes in the muscles. A partial reaction of degeneration is the type of change most commonly found. The nerves react to both faradic and galvanic currents, but there is a progressive diminution in the degree of contraction, and hence very strong currents are necessary in order to produce any effect. The muscles respond to the galvanic current very sluggishly, with a so-called "vermiform contraction," and usually react better to the positive than to the negative pole. Finally, all faradic reaction is lost, a complete reaction of degeneration develops, and as the muscle becomes completely atrophied all reaction to galvanism is lost. The knee-jerk is not affected in the disease until the rectus femoris becomes atrophied, when it becomes gradually diminished, and finally is lost. As already stated, sensation is normal from beginning to end, and there is no affection of the sphincters.

FIG. 68.



Atrophy of the muscles about the shoulder-blades and arms in a case of chronic anterior poliomyelitis. The triceps and latissimus dorsi have escaped.

The duration of the disease varies very much in different cases. The most rapid case that I have seen was two years in its duration. But an arrest of the atrophy may occur at any time in the course of the case, and hence the prognosis must be very guarded, as it is unjust to condemn these patients when there is still hope.

While the majority of the cases correspond to one of the three types described, it is not to be forgotten that a progressive muscular atrophy may begin in any muscle of the body and advance to any other set of muscles with a most irregular course. Thus Werdnig[1] has reported a case in which the paralysis began in the muscles of the back and glutei, then advanced to the neck and throat, and finally to the extremities. Strümpell[2] has recorded a case in which the progress was from the hands to the forearms and then to the shoulder, in distinction from the ordinary progress from the hands to the shoulders. Others have described cases in which both hands and legs have been invaded together.

Diagnosis. As already stated, chronic atrophic paralysis may occur in several diseases. The differentiation of chronic anterior poliomyelitis from amyotrophic lateral sclerosis is as follows:

In amyotrophic lateral sclerosis there is an increase in the knee-jerks early in the disease, an early development of Babinski's reflex —*i. e.*, retraction of the great toe and flexion of the other toes on tickling the sole—and increased mechanical excitability in the muscles that are paralyzed; a tendency to rigidity in the movement of the legs, and some spastic rigidity of the arms; also, an increase in the tendon reflex at the elbow and wrist. In other words, the symptoms of lateral sclerosis are added to the symptoms of progressive muscular atrophy, and it is by a discovery of these additional symptoms that the diagnosis is made. The progress of the case is usually more rapid in amyotrophic lateral sclerosis than in chronic anterior poliomyelitis; and bulbar symptoms either appear early in the affection or precede the paralysis in the limbs.

A differential diagnosis from muscular dystrophy is to be made by a consideration of the progress of the case and the succession of implication of the various muscles, which is quite characteristic in the different types of muscular dystrophy, and differs from that already described. (See Muscular Dystrophy, Chapter XIV.) In muscular dystrophy there is never any fibrillary contraction in the paralyzed muscles. The atrophy of the muscles is often attended by a deposit of fat, so that there is an appearance of hypertrophy in the muscles that are really atrophied. The tendon reflexes in muscular dystrophy are progressively diminished as the muscles become atrophied. The history of the case, its onset in childhood, its development in a child of a family in which other members have been known to be affected are important facts in connection with the differentiation from progressive muscular atrophy. There is no electrical change in the muscles affected in dystrophy.

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[1] Arch. f. Psych., xxvi., 706.

[2] Deut. Zeitschr. für Nervenheilk., iii., 6.

In the muscular atrophies that are consequent upon neuritis there are usually sensory symptoms and tenderness along the nerve trunks, and there is a history of one of the well-known causes. (See Chapter III.)

In cases of paralysis of the ulnar nerve the distribution of the paralysis and atrophy to the muscles in the hand may simulate progressive muscular atrophy, but it is to be remembered that in ulnar paralysis the first and second interossei escape and there is usually some affection of sensibility in the little finger (see page 69).

The symptoms of progressive muscular atrophy are sometimes the first symptoms to develop in a case of syringomyelia, but the subsequent appearance of dissociated anæsthesia (a loss of sensibility to pain and temperature, while that to touch is present) and of trophic symptoms, ulcerations of the skin, etc., together with the usual increase of the knee-jerks, will enable the diagnosis to be made. (See Chapter XV.)

A differentiation between progressive muscular atrophy and the Charcot[1]-Marie-Tooth[2] type of atrophic paralysis is somewhat difficult. That disease appears in early youth. It produces a progressive muscular atrophy of the peroneal type in the legs and of the Aran-Duchenne type in the hands, about simultaneously or in rapid succession; but the paralysis does not extend, as a rule, above the knees or elbows, though occasionally the muscles around the thigh and glutei are invaded. The contrast between the atrophied lower parts of the limbs and the well-developed upper portions of the limbs is very marked. Fibrillary tremor and reaction of degeneration are present in both diseases, but in the Charcot-Marie-Tooth type sensation is usually diminished in the legs and feet and on the hands. In this disease a few autopsies (Hoffman, Muhlenburg, Dubreihl, and Marinesco) have demonstrated a peripheral neuritis with some posterior sclerosis, but no affection of the anterior horns. (See Chapter XIV.)

Prognosis. The prognosis in chronic anterior poliomyelitis is a serious one, as the tendency of the disease is to go on to extreme atrophy and death from respiratory or bulbar paralysis. It must be remembered, however, that an arrest at any time in the course of the case is possible. Gowers maintains that the cases in which the disease begins on both sides simultaneously are more liable to a spontaneous arrest. When the disease has once been arrested there is no return of power and no increase in size in the affected muscles, which are permanently paralyzed. If the disease starts up a second time after an arrest of its progress the prognosis is most unfavorable, as a rapid course is probable. Death occurs from paralysis of the respiratory muscles, or from bronchitis or pneumonia consequent upon the disturbance of respiration, or from bulbar palsy.

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[1] Rev. de Méd., February, 1886, p. 97.

[2] Tooth. Dissertation, London, 1886. Neurol. Centralbl., 1887, vi. 16.

Treatment. The treatment of chronic anterior poliomyelitis is the same as that of acute anterior poliomyelitis in the chronic stage—namely, general hygienic measures; good food, especially of fatty kind; fresh air, and every means of keeping up the general nutrition of the patient. Exercise, while not prohibited, should be advised in great moderation, and, if the legs are affected, walking should be avoided, so as not to put any strain upon the paralyzed muscles. Any effort which causes fatigue is sure to be followed by an increase in the weakness and a more rapid atrophy. Any exhausting disease, such as an intercurrent attack of the grippe, pneumonia, or any infectious fever, or a severe gastro-enteritis is very likely to increase the paralytic symptoms. Thus in one of my patients who had been in a stationary condition for a year an attack of the grippe was followed by a rapid increase in his paralysis. There is no treatment that will arrest the progress of the paralysis. Massage and rubbing are usually prescribed and may possibly aid in keeping up the general strength by increasing the nutrition of the atrophied muscles. I have never seen any benefit from the continued use of electricity, though it is usually prescribed. If it is used it should be with a current only sufficient to produce contraction in the muscles, and very strong or very many long-continued applications are to be avoided on the same principle that exercise is to be avoided. The muscles should not be overworked or fatigued. In one case the use of mechanical massage and vibratory treatment at the Zander Institute caused marked improvement and was followed by an arrest in the progress of the disease for a year.

General tonics are of distinct service, and I have seen temporary improvement from the employment of strychnine, $\frac{1}{50}$ grain, three times a day for four days in the week, alternating with arsenic, $\frac{1}{50}$ grain three times a day for the other three days of the week. Some authors recommend the hypodermic use of strychnine, but in a long-continued chronic disease of this kind very little advantage is to be obtained, and the dangers attendant upon septic infection are many. Apparatus may prevent deformities and assist weakened joints to bear the weight of the body after walking becomes impossible.

CHAPTER XIII.

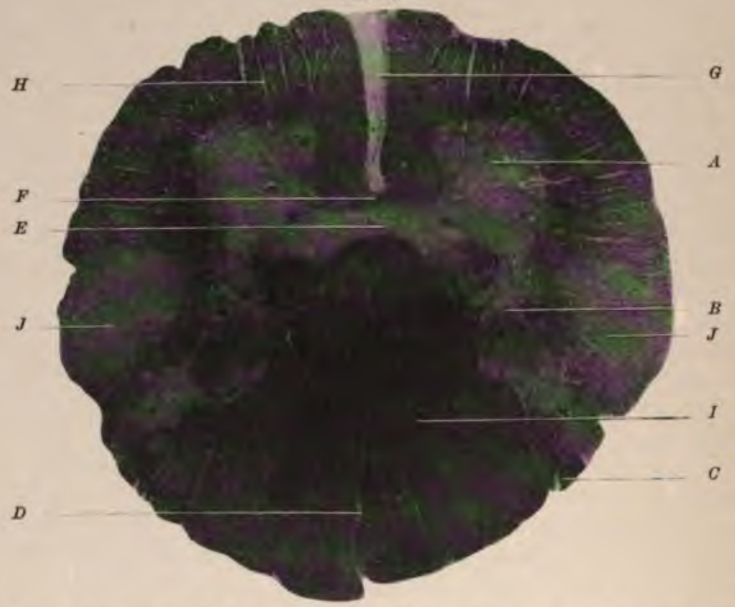
AMYOTROPHIC LATERAL SCLEROSIS.

THIS is a chronic progressive form of spinal paralysis characterized by the symptoms of progressive muscular atrophy in the arms and of lateral sclerosis or spastic paraplegia in the legs. It was first described by Charcot in 1872 and was carefully studied by his pupils, Gombault in 1877 and Debove in 1879. It has been called Charcot's disease. But while Charcot believed that it was a disease of the spinal cord only, it is now known that all the motor elements of the nervous system, from the cortex of the brain to the termination of the nerves in the feet, are involved, both the cortico-spinal and spinomuscular elements being simultaneously affected.

Pathology. The pathological condition consists of, first, a gradual progressive atrophy in the motor neurones lying in the anterior horns of the spinal cord, similar to that described as occurring in chronic anterior poliomyelitis. This is sometimes limited to the cervical region, but in long-continued cases the lumbar region is affected, and in some cases the entire cord is involved. These changes in the cells are also present in the motor nuclei of the cranial nerves in the medulla and pons. Secondly, in addition to these changes in the spinomuscular element, there are atrophy and degeneration in the cortico-spinal element which lies in the lateral pyramidal, and anterior median columns of the spinal cord. These changes have been followed upward through the medulla, pons, crus, and internal capsule to the motor cells of the brain. And recent investigation has shown that in the majority of cases of advanced amyotrophic lateral sclerosis there is a degeneration, with atrophy of the larger cells of the second and third layers of the cortex about the fissure of Rolando, which are the neurone bodies of the cortico-spinal elements of the motor system. This degeneration of the cortico-spinal elements begins in the ends of the axones in the spinal cord and advances upward until the entire neurone, of whatever length, becomes affected and atrophied. The sclerosis is a replacement hyperplasia in the spinal cord, the increased connective tissue taking the place of the atrophied nervous elements. The sclerosis is not wholly limited, however, to the motor tracts in the cord, but is present throughout the antero-lateral columns in the vicinity of the gray horns, as in chronic anterior poliomyelitis. (See Fig. 64.) It is evident, therefore, that the association tracts belonging to the motor system of the cord are involved as well as the longer voluntary tracts; hence this disease is one of the most widespread of all the so-called "system diseases" of the spinal cord.

The point of beginning of the pathological process varies in different cases. In some cases it is evident that the lesion begins in the cervical region of the spinal cord in the gray matter, and hence the symptoms first appear in the muscular system of the arms. This has been the course in about 60 per cent. of my cases. In other cases the lesion commences in the lateral columns of the cord, and then the spastic paralysis of the legs is the first evidence of the disease. This is the second form and has been the history in 30 per cent. of my cases. In either case in the course of the disease both sets of symptoms appear. In the cases that begin in the spinal cord the tendency of the disease is to extend to the medulla and pons, so that symptoms of bulbar paralysis ensue and terminate the case. In other cases, however, bulbar paralysis is the first disease to appear, and later on, after its symptoms are well marked, the symptoms of spastic paraplegia or of progressive muscular atrophy in the hands indicates that the original disease was not simply bulbar palsy, but amyotrophic lateral sclerosis. This has been the course in 10 per cent. of my cases. The lesion is a slowly progressive one, occasion-

FIG. 64.

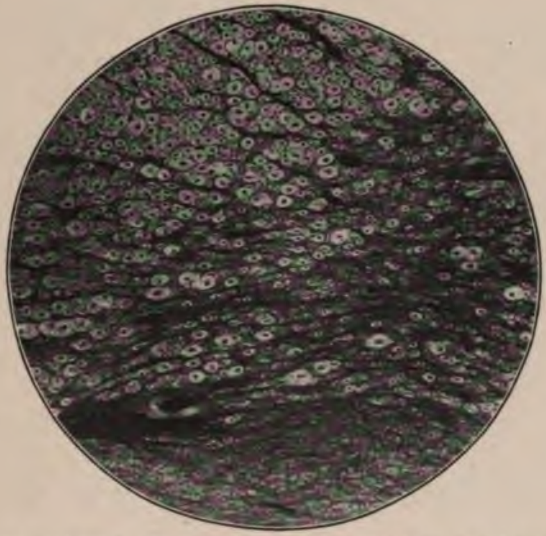


The lesions in amyotrophic lateral sclerosis, lumbar region. *A*, anterior horn, atrophied and sclerotic, no cells remaining; *B*, posterior horn; *C*, posterior nerve root; *D*, posterior septum; *E*, posterior commissure; *F*, anterior commissure; *G*, anterior fissure; *H*, antero-lateral column, slightly sclerotic; *I*, posterior column; *J*, lateral pyramidal tract, sclerotic. (Blocq.)

ally comes to a standstill for a time, but never has any tendency to recover, regeneration not appearing in the degenerated neurones.

Etiology. Very little is known about the causation of this disease. It develops in persons between the ages of thirty-five and fifty years. Cold, overexertion, various forms of poisoning, either by alcohol, arsenic, lead, mercury, or by toxins of infectious diseases, or from auto-intoxication through disturbance of the gastro-intestinal tract, rheumatism, gout, and diabetes have all been mentioned as causes without any absolute proof. There is no proof that the affection develops subsequently to syphilis. Gowers has seen it develop after severe injuries. A probable theory of the affection is that in certain families a congenital weakness of the motor elements of the central nervous system is present, a fact which receives some support from the occurrence of the disease occasionally in several members of the same family. In some cases an extensive endarteritis has been found in the spinal bloodvessels, and this has been supposed to be the cause in these cases.

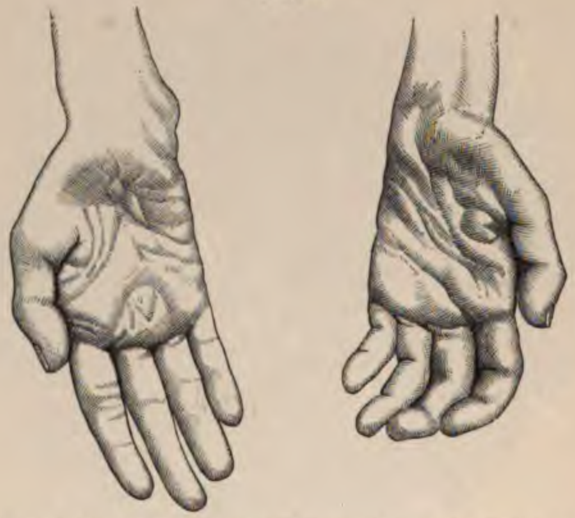
FIG. 65.



Sclerosis of the spinal cord. The specimen is taken from the border of the lateral pyramidal tract in a case of amyotrophic lateral sclerosis. The upper part of the specimen is normal; the lower part is sclerotic, and in this part few or no fibres remain.

Symptoms. Amyotrophic lateral sclerosis usually begins with stiffness in the muscles and increased reflex excitability, soon followed by atrophy and paralysis in one or both hands, the small muscles of the hands being the parts first affected, as in progressive muscular atrophy of the Aran-Duchenne type. The paralysis then advances to the forearms, though occasionally it appears in the muscles about the shoulders before those of the arms are invaded. Paralysis and atrophy go hand-in-hand, and the weakness is proportionate to the size of the muscle, as in progressive muscular atrophy. Fibrillary contractions are present in the muscles from the beginning of the disease, and may be elicited by exposure to cold or by percussion. The order in which the various muscles are invaded corresponds quite closely to that described in progressive muscular atrophy of the Aran-Duchenne type. When all the symptoms are well developed in the upper extremities the arms are adducted to the body, the forearms are pronated, and the hands flexed or in the position of *main en griffe*. There is some resistance offered to passive movements, as the muscles are rigid and spastic even when quite weak.

FIG. 66.



Extreme atrophy of the thenar, hypothenar, and interossei muscles of the hands in amyotrophic lateral sclerosis. (Dejerine.)

The symptoms appearing in the legs may follow or precede those in the arms; but, as a rule, a considerable length of time (several months) intervenes between the two. When the disease begins in the legs the early diagnosis is lateral sclerosis. The legs become stiff, and the muscles rigid, so that the patient moves them with some difficulty; the knee-jerks are increased, and ankle clonus and the Babinski reflex can be elicited. The gait becomes a true spastic gait, with short steps, scraping of the foot upon the ground, tendency to overlapping of the knees and feet, and great rigidity in bending of all the joints. The muscles of the legs do not atrophy in the early stage and are not the subject of fibrillary twitches. The symptoms may not begin in both legs at the same time; in fact, one leg may be quite rigid for several months before the other is attacked. The rigidity gives rise to some aching and discomfort in the muscles, but there are no sharp pains, and there are no sensory disturbances either of the nature of anæsthesia or paræsthesia. The bladder and rectum are not in any way affected.

In this condition of partial paralysis the patient may remain for several months, the disease coming to a standstill, or the disease may go on progressively. One patient in my clinic remained in a stationary state for four years after all these symptoms had developed. If it progresses the spastic rigidity of the legs becomes so intense that walking is impossible, and the patient is confined to the bed. Then the muscles which have hitherto been rigid begin to atrophy, and to show fibrillary twitchings, and the spastic paraplegia is succeeded by a flaccid paralysis quite similar to that which has appeared from the outset in the hands and arms.

Sometimes the paralysis extends from the upper extremities to the neck, the head falls forward, the chin resting on the sternum, and the head cannot be raised or turned.

Some months before complete paralysis has developed in the arms it is common to observe some symptoms of bulbar palsy. A few cases begin with these symptoms, and only develop paralysis of the arms and legs later. The first symptom noticed is a disturbance of speech due to a paralysis of the muscles of the tongue, lips, mouth, and palate. The speech is slower, pronunciation being difficult; the voice becomes monotonous, possibly nasal and indistinct, and finally pronunciation of both linguals and labials is so imperfect that it is with difficulty that the patient is understood. As this paralysis goes on the tongue becomes thin, is thrown into folds, and shows fibrillary twitchings, and cannot be freely moved and protruded from the mouth. The palate also becomes paralyzed, the uvula is no longer properly elevated, and hence swallowing is imperfect, fluids return through the nose, and attacks of choking are frequent. The face also gradually becomes paralyzed, especially in its lower portions, it is flattened so that there is a mask-like expression, and gradually all expression is obliterated. Whistling, blowing, or kissing is impossible. The mouth is partly open, the saliva runs from the corners, and the act of chewing is interfered with on account of the paralysis of the cheeks and on account of the weakness of the muscles of mastication. The muscles of the face may show some fibrillary twitching for some time before they become paralyzed. Finally, the upper branches of the facial nerve are affected, patients cannot close the eyes, but the ocular muscles do not often share in the paralysis. One or two cases have been reported in which the Argyll-Robertson pupil has been present.[1] The inability to swallow may cause death from choking or from respiratory paralysis, or a pneumonia may develop from irritation of the lung by particles of food which are inhaled.

One of the characteristic symptoms of the disease is the great increase in the muscular irritability in all the muscles that are affected. Percussion, either on the muscle or on its tendon, causes a quick, unusually sharp contraction, and this exaggeration of reflex activity throughout the entire body, both in the muscles that are atrophic and in those that are spastic, is characteristic of the disease, and is not present in progressive muscular atrophy. Percussion of almost any point upon the limbs—on the muscle, tendons, or the periosteum—produces sudden contraction of groups of muscles, and sudden extension of any tendon may be followed by a clonus. Percussion of the facial muscles, especially of the masseter, and of the jaw produces sudden contractions and even the chin reflex may be so exaggerated as to give rise to a clonus.

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[1] **Schlesinger.** Obersteiner's Arbeiten, 1900, vol. vii. p. 154. Zür Kenntniss atypische Formen der Amyotrophischen Lateralsclerose.

The atrophic paralysis is usually attended by cramps and also by deformities (*main en griffe* or drop-wrist) such as appear in progressive muscular atrophy. If deformities occur in the leg talipes may develop from contracture of the posterior tibial tendons.

In the later stages of the disease the muscles of the trunk and shoulders and neck may become atrophied, so that sitting up or lifting the head is impossible.

The electrical examination shows a diminution of excitability both to faradism and galvanism, the muscles that are atrophied requiring a stronger current for the production of contraction. A partial reaction of degeneration may also be found.

Tachycardia occasionally develops in the later stage of the disease, as in bulbar palsy, and usually is an exceedingly serious symptom, as it may cause death. The brain is not in any way affected, excepting in its motor region. Intelligence is preserved, consciousness is not affected, memory is not impaired, and epileptic attacks do not occur. Occasionally, however, as in bulbar palsy, an unusual emotional excitability develops, and causeless crying or laughing indicate a weakening of the patient's power of self-control.

The duration of the disease varies between two and ten years, depending entirely upon the time of onset of the bulbar symptoms, which are the immediate cause of death. If these symptoms appear early or are the first to occur the patient rarely lives more than two years. If the symptoms first appearing are those of lateral sclerosis the case may last for many years. The immediate cause of death is usually disturbance of respiration or suffocation from food getting into the larynx, or pneumonia from food being received in the lungs, or from heart failure.

The course of the disease when it begins with bulbar palsy is illustrated by the following case: I. D., aged forty-two years, had been much exposed to wet, to extreme heat, and to cold in his occupation, and had suffered from muscular rheumatism for years. In February, 1889, he noticed double vision, due to a weakness of the left external rectus muscle, and ptosis which first affected the left eye and then the right eye. The ptosis gradually subsided, but the strabismus remained. In March, 1889, his speech became thick, and in

April he began to have difficulty in swallowing, fluid food coming out of his nose. In May he noticed difficulty in chewing, and his friends saw a change in his facial expression due to a weakness of all the facial muscles. During all this time he felt an increasing weakness in all his movements. His neck had become stiff and his head tended to fall forward and was held with chin projecting beyond the line of the body. On several occasions after March, 1889, he had fainting attacks in which he became pulseless and pale. In June, when I saw him, he was thin and pale, clear in his mind, but feeble in all movements. His eyes were both turned in a little and his left eye looked up. He had corresponding double images, but no nystagmus and his pupils were normal. His optic nerves were normal. There was anosmia on the left side. His sensation in the face was normal, but the muscles of mastication were atrophied, did not react to faradism, and were so weak that chewing was impossible. He moved his jaw with his hand and gave it constant support. His face was expressionless; he could not whistle; food collected in his cheeks, but all his facial muscles could be slightly moved, and reacted to faradism. His palate was paralyzed, and it was to this cause that his defect of speech was due, as the tongue was not paralyzed or atrophied. His neck muscles were weak. There was an atrophic condition of the thenar muscles and interossei in the hands, and marked weakness in all the muscles of the forearm. The deltoid and muscles of the arms were in good condition. His gait was slow and feeble, but not spastic; but his knee-jerks were exaggerated and ankle clonus was obtained. He controlled his sphincters well. During the following year his paralysis increased in his arms and legs, his inability to talk became more apparent, and in August, 1890, he choked to death.

In the following case the symptoms began in the leg on one side: A woman, aged forty-five years, began to suffer from weakness, fibrillary twitchings, and atrophy in the right leg, with reaction of degeneration in the muscles supplied by the peroneal nerve, in February, 1890. This was supposed to be due to traumatism of the nerve, as she had had an injury. But in April, 1890, an atrophy had begun in the thenar and hypothenar eminences of the right hand. These symptoms increased, and in October, 1890, she was almost completely paralyzed in the entire right side and partially in the left side, there being everywhere an atrophic state of the muscles with fibrillary contractions and reaction of degeneration. There was no pain, no sensory disturbance, no bladder or rectal symptoms. The tendon reflexes were increased. During the winter of 1890–1891 the paralysis extended, and she was finally confined to the bed. In April, 1891, the muscles of her throat and face were affected, and in July she died of respiratory paralysis.

Diagnosis. The diagnosis of this affection does not present any difficulties. The muscular dystrophies are not attended by fibrillary twitchings or by increased tendon reflexes or by bulbar symptoms.

Bulbar paralysis is not, as a rule, attended by any spinal symptoms or paralysis, or increased rigidity, or increased knee-jerks. If such symptoms develop in the course of bulbar paralysis it is evident that the disease is a true amyotrophic lateral sclerosis in which the bulbar symptoms have been the first to appear. Compression of the cervical portion of the spinal cord by tumor or by pachymeningitis is usually attended by pain in the shoulders, neck, and arms, by stiffness of the cervical vertebræ, and pain from pressure. Localized compression of the spinal cord, such as occurs in these diseases, if attended by spastic paraplegia is usually attended also by sensory disturbances and by an affection of the bladder and rectum. The diagnosis from syringomyelia is made by the absence of sensory disturbances or trophic affections, and, as is well known, in this disease bulbar symptoms, while occasionally developing, are unilateral and do not present the typical features of a bulbar palsy. Tumors of the spinal cord of the nature of gliomatosis give rise to sensory symptoms as well as to spastic paraplegia and progressive muscular atrophy. The same is true of chronic myelitis. In any case of primary lateral sclerosis it must be remembered that spastic paraplegia often goes on to atrophic paralysis. Hence many cases of true amyotrophic lateral sclerosis beginning with symptoms of spastic paralysis in the legs are supposed to be primary lateral sclerosis. Time alone can decide between the two affections. In a large number of cases diagnosticated as lateral sclerosis the lesions of amyotrophic lateral sclerosis have been found.

Treatment. The only treatment is general tonic treatment. It is well to prevent all strains and exertion and to build up the general health in every way possible. Massage is to be used and electrical applications, but with the same caution against over-fatigue of the muscles by these measures as has been mentioned in connection with the treatment of chronic anterior poliomyelitis. Long-continued warm baths and douches without very great alternations of temperature are of considerable service in maintaining the nutrition. When the bulbar symptoms appear the patient should be instructed in the art of passing a tube into the stomach, so that as soon as difficulty of swallowing appears nourishment may be carried on in this manner, and thus the danger of suffocation may be avoided. The use of strychnine does not appear to retard the progress of the disease, and, in fact, is objectionable on account of the tendency to spastic paraplegia.

CHAPTER XIV.

MUSCULAR DYSTROPHIES.

The Classification of Various Forms. Etiology. Pathology. Pseudomuscular Hypertrophy. Erb's Juvenile Form of Dystrophy. Landouzy-Dejerine Form of Dystrophy. Muscular Atrophy of the Peroneal Type. Myotonia Congenita, Thomsen's Disease.

THERE are certain forms of paralysis appearing, as a rule, in early life that are due to muscular and not to nervous disease. These have been named the muscular dystrophies. The muscles appear to be affected primarily by a progressive atrophy. This atrophy involves the fine muscular filaments and proceeds slowly until in the end even the largest muscles may be reduced to a small band of connective tissue. In some cases there occurs a deposit of fat around the atrophied muscle fibres, filling up the empty spaces within the muscle so that no apparent atrophy occurs. In some cases this deposit of fat is abnormally great, increasing the size of the muscle until it may be two or three times its normal diameter, and producing the appearance of a large muscle where there is really an atrophied muscle. This form is termed pseudomuscular hypertrophy. In other cases there is a true hypertrophy of the muscle fibres. Inasmuch as any form of muscular dystrophy produces at first paresis and then paralysis, it is very natural that these muscular dystrophies should be mistaken for spinal paralyses, though a brief study of their characteristic features will make it clear that no such mistake is warranted.

The muscular dystrophies have been classified by Erb,[1] who has gathered and analyzed all the facts in regard to this affection, more completely than any other author. Erb distinguished first between cases occurring in infants and those developing in adult life, though this distinction appears to be of little moment, inasmuch as an analysis of a large number of cases demonstrates that the disease may develop at any age. It must be admitted, however, that a very large proportion of the cases appear in infancy or childhood, and that when adults are affected the disease usually begins before the twentieth year. Erb further classified these cases, first into those in which there is an hypertrophy of muscular tissue, either a pseudohypertrophy or a true hypertrophy of the muscle fibres, and, secondly, those in which there is a progressive atrophy without any deposit of fat. This classification, while of value clinically, appears to rest upon no essential pathological factor, for all grades in the process are possible. In some cases the deposit of fat is excessive, causing pseudohypertrophy; in other cases there is a deposit of fat taking the place of atrophied muscle, but without producing any apparent change in the size of the muscle, and in many cases of extreme atrophy of the muscles fatty deposits are found between the muscle fibres; hence this classification does not rest upon a pathological basis. And we may find all these conditions in different muscles in the same patient.

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[1] Deut. Zeitschr. f. Nervenhk., i., 13 and 173.

A further classification has been based upon the distribution of the affection in different muscles. Here, unfortunately, the different types have been named after the observers who happen to have first carefully described them. Thus we have the Duchenne type of pseudohypertrophy, and we have the Erb type of juvenile dystrophy, in which the muscles about the shoulder-blades and arms are chiefly affected. We have the Sachs-Hoffman type, in which the affection begins in the peroneal muscles of the legs. We have the Landouzy-Dejerine type, in which the muscles of the face and arms are affected, and we have the Charcot-Marie-Tooth type, in which legs and forearms and back are affected, thighs and arms and face escaping. It is true that the various cases observed conform in many particulars to one of these different types, but there is no essential difference between them in the causation or in the pathology. And intermediate types have been observed not conforming to any of these. Furthermore, it is found that one type may progress and gradually merge into another. It is evident, therefore, that these attempts at classification in the discussion of muscular dystrophies are not fully satisfactory.

Etiology. The causation of muscular dystrophy is a matter of considerable interest. The disease is undoubtedly a disease of development, and from its marked tendency to appear in very early life, to attain a certain status, and then to be arrested without any tendency to repair, or else to progress steadily until it causes an entire destruction of the muscular system, it seems probable that it must be traced to an inherent defect of development in the muscles affected. It seems as if these muscles were not endowed with the proper vitality, and, after a short life, wither and die.

We have evidence in other organs of the body of this difference between the life-period of a special structure and that of the entire body. Thus the life-period of the first and even of the second teeth is a comparatively short one as related to the life-period of the individual. The life-period of the hair, of the reproductive system, of the eye and ear, and of the brain may be less than that of the entire individual. We see in the study of optic atrophy and of auditory atrophy that there are some families in which these diseases develop, even in early life, without apparent cause, the only explanation being that these nerves have not the power of survival which other nerves have. We recognize the existence of senile atrophy of the brain in old age, but we must admit that the term "old age" cannot be fixed within definite limitations, for some individuals become old at the age of sixty years, while others are young and vigorous at the age of eighty years. I have now under my observation a lady of the age of ninety-nine years, all of whose visceral functions are as vigorous and active as they were at the age of seventy, but whose brain for the past ten years has been practically dead, powers of perception in eye and ear being suspended, powers of memory being almost obliterated, and powers of reasoning being reduced to the level of a child of three years. Thus, it is evident that the organism as a whole may outlive any one of several of its organs, provided those organs are not essential to life. And the duration of vitality of any organ varies greatly in different individuals. This fact is particularly evident in regard to the reproductive system both in males and females, and it is equally apparent in regard to the muscular system. For it is a matter of comment that the muscles of old people wither and become feeble long before their death.[1]

It is quite evident from the consideration of these facts that the explanation of muscular dystrophies is to be found in an inherent inability of survival of certain muscles in certain individuals. In other words, that the disease is one of congenital defective power of evolution. This explains to a certain extent the frequency with which we find that several members of a family are affected by this type of disease. It is true that isolated cases are not uncommonly met with where careful investigation fails to reveal any member of the family or any member of any collateral branch similarly affected. But in the majority of cases it is found that if the patient has no brother or sister similarly diseased, or has not inherited it directly from either parent, he will have several cousins who have suffered, possibly several second cousins who have suffered, and it is not uncommon to be able to trace the disease through four or even five generations.

Whether extraneous causes, such as undue muscular effort, exposure to cold, the unfavorable effects of the occurrence of infectious diseases, falls, and blows, have anything to do with setting up the disease in those who have an inherent tendency toward weakness of the muscles is a matter which may be open to doubt, though these factors have been mentioned as exerting a causative agency.

Pathology. The changes found in the muscles in this disease are those of a simple progressive atrophy. It will be remembered that the muscle fibre, which may be 5 cm. long and from 15 to 55 mm. broad, has a striated appearance due to the existence of two types of structure within its substance, one of which interrupts the light more completely than the other. Each muscle fibre is surrounded by a sheath of connective tissue (the sarcolemma) within which lie nuclei or long oval cells from which the muscle fibre itself has developed. These muscle fibres, surrounded by the sarcolemma, are really made up of fibrils of very fine structure, each fibril originating from a single cell, and the fibrils are separated from one another by a structure termed the sarcoplasma; hence, under the microscope a muscle fibre has a double striation, a transverse striation, and the longitudinal fibrillar structure. These fibres are gathered into greater or lesser bundles, forming the muscle, and they are supplied by nerves whose terminal filaments spread out upon the surface of the fibre in the so-called motor plate. Within the muscle and within the fibres lie oval bodies called the muscle spindles which contain the sensory filaments through which the muscular sense is received. (See Fig. 7, page 24.)

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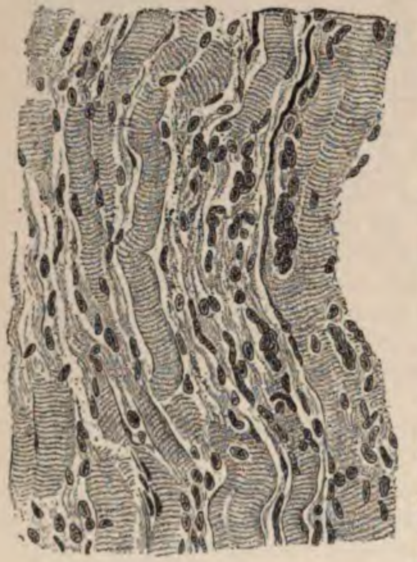
[1] See Gowers: Abiotrophy. London, 1903. Published after this chapter was in type.

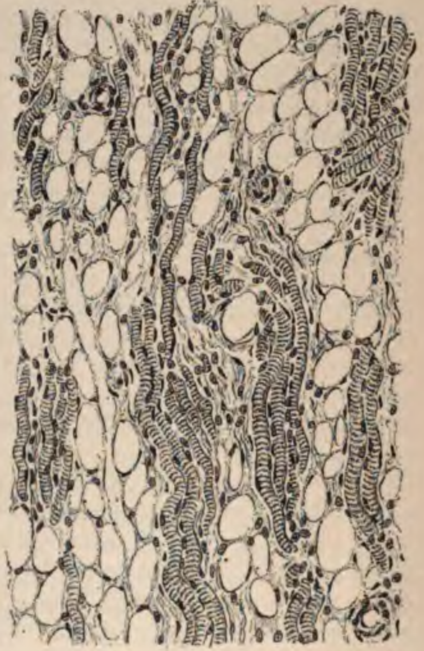
When a muscle undergoes atrophy we may find, first, a simple reduction in the calibre of the individual fibres. (Fig. 67.) There is a thinning in the entire fibre, which becomes narrower and shorter, preserving until the end its striated appearance, though it becomes pale and sometimes a little pigmented by the deposit of coloring matter.

FIG. 67.



Atrophy of a muscle, with increase of nuclei. × 250. (Schmaus.)

FIG. 68.



Atrophy of muscle, with deposit of fat about the atrophied fibres. × 250. (Schmaus.)

Secondly, in other cases there is a degeneration of the muscle, consisting of a swelling or œdema, followed by an albuminoid or fatty degeneration of the muscle fibre, resulting in the deposit of small fatty globules and granular masses within the sarcolemma. As the muscle becomes degenerated the individual fibre contracts, obliterating the striated appearance, leaving the muscle with a homogeneous, smooth, or yellow tint. As the process goes on all trace of the muscular substance may vanish, leaving the sarcolemma containing only fatty globules. Sometimes waxy deposits may occur within the muscle, with compression and obliteration of the fibres, but this is not common in the dystrophies. Occasionally as the muscle undergoes progressive atrophy its sheath is filled up by an œdematous exudation. But usually there is merely an increase in the cells of the sarcolemma. Under some circumstances there is not only within these empty sarcolemma sheaths a fatty remainder of the degenerated muscle, but between the sheaths there is an actual deposit of fat of new formation. (See Fig. 68.) Under these circumstances not only is the space formerly occupied by the normal muscle filled out by fat, but the fatty deposit may increase the actual diameter of the muscle; and if this goes on to any extent pseudohypertrophy of the muscle will be presented. Along with the fatty deposit there is usually an hypertrophy of the connective tissue in the muscle, so that when the process becomes extreme the muscle is turned into a mass of fat separated by trabeculæ of connective tissue. If, after the course of time, the fat is reduced in volume and is finally absorbed the muscle will have as its only relic this connective-tissue strand, and as this contracts longitudinally it will cause a shortening of the atrophied muscles.

Thirdly, along with the atrophy we find occasionally a true hypertrophy of individual muscle fibres. The fibrils are increased by true formation of muscle tissue from their nuclei within the sarcolemma, and not only increased in number, but also increased in lateral diameter, so that giant muscle cells and giant muscle fibres quite similar in structure to a normal fibre are evident. Any of these processes may go on alone or may be combined with others in the process of muscular dystrophy. The connective-tissue changes which were for a time thought to be primary have long been known to be of a secondary nature, a hyperplasia of connective tissue within the muscle occurring just as a hyperplasia of neuroglia occurs in the spinal cord to take the place of atrophied substance. In the connective tissue near to the ends of the muscle new formations of thick tendons may be seen.

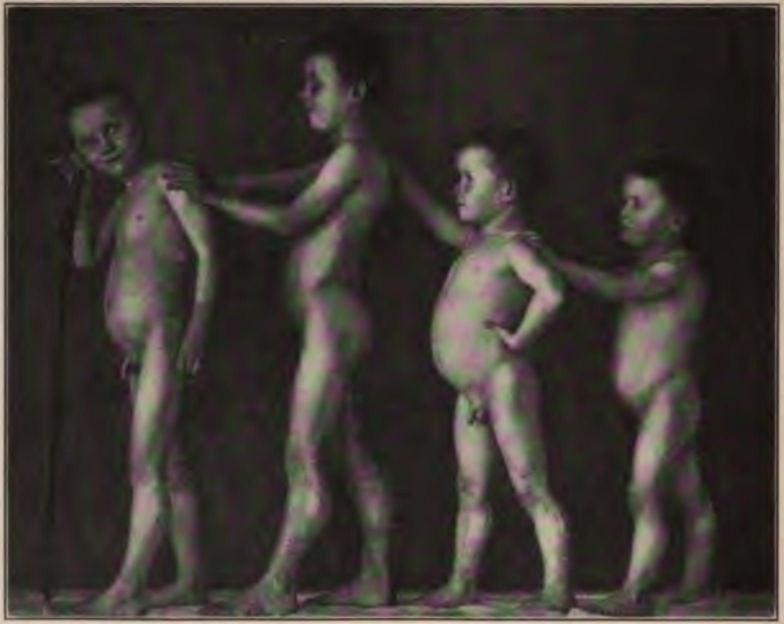
These changes in the muscles are found in all forms of muscular atrophy both spinal and peripheral. It is thought that in the dystrophies the development of hypertrophy of the muscle fibre in connection with the atrophy is a peculiar feature of the disease. It is not often that deposits of fat are found within the muscles in the spinal and peripheral types of atrophy.

Symptoms. From the clinical standpoint we recognize the following types of the disease, the symptoms of which differ distinctly from one another:

1. Pseudomuscular hypertrophy of Duchenne.
2. Erb's juvenile dystrophy.
3. Landouzy-Dejerine type of dystrophy.

1. **Pseudomuscular Hypertrophy.** This disease begins in children between the second and the seventh year, and has often made considerable progress before it is brought to the attention of the physician. It begins by a gradual increase in the size of the calves of the legs, which are at first subjects of pride to the mother, but soon awaken her apprehension because of being out of proportion to the growth of the rest of the limbs and because they appear to be attended by a certain clumsiness of gait. The child, in learning to walk or in walking, begins to stumble, to go up stairs with some difficulty, to get tired too soon after walking or running, and to walk with a peculiar gait. This gait is characterized by a dragging of the legs, which appear to be raised from the ground with difficulty, and in order to drag the leg the child throws its body from side to side and raises its pelvis in the act of walking. As the disease progresses great difficulty is experienced in rising from a chair or in getting up from the floor, and these children soon learn to aid themselves by the use of their hands, which give support where the muscles of the legs

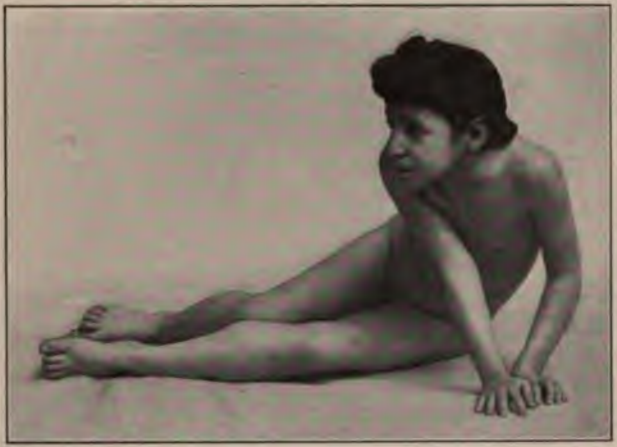
FIG. 69.



Pseudohypertrophic muscular dystrophy. Four brothers, aged twelve, eleven, eight, and seven years. The calves and the anterior surface of the thighs are hypertrophied. The muscles of the back are atrophied. The eldest has so much weakness of the muscles of the neck that he cannot hold up his head. (Curschmann, Klin. Abbildungen.)

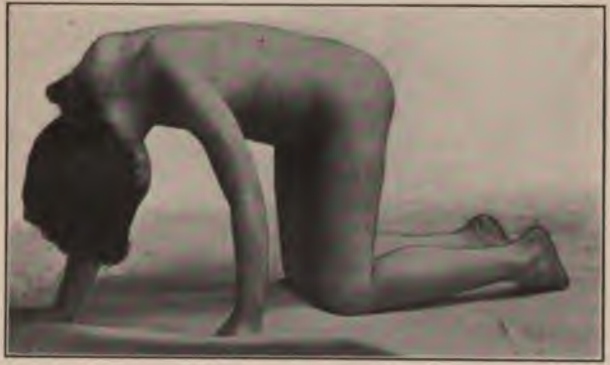
are weak. When the disease is well advanced these children rise from a lying to a standing posture in a peculiar manner that is characteristic of weakness in the muscles of the calves, quadriceps femoris, and glutei muscles and muscles of the back (Figs. 70 to 74). This typical method of getting on the feet, together with the peculiar waddling gait, is sufficient to make a diagnosis of the affection. When the disease is fully developed inspection shows an hypertrophy of the muscles of the calves (Figs. 75 and 76), an hypertrophy of the anterior

FIG. 70.



Pseudohypertrophic paralysis. The act of rising.

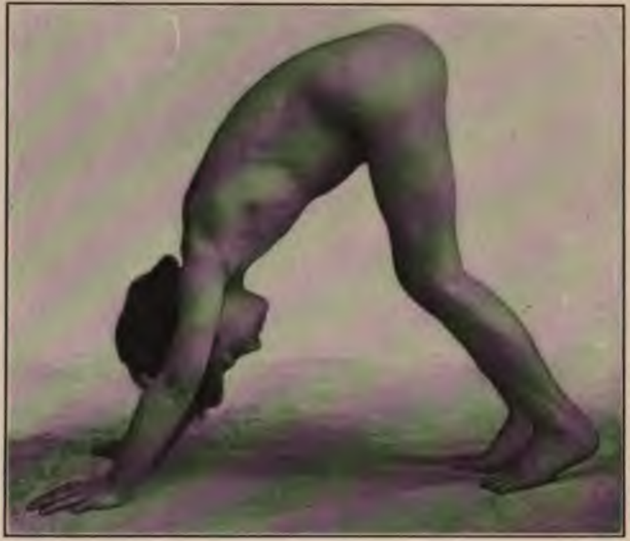
FIG. 71.



Pseudohypertrophic paralysis. The act of rising. This position shows the weakness of the muscles of the neck and the atrophy of the arms.

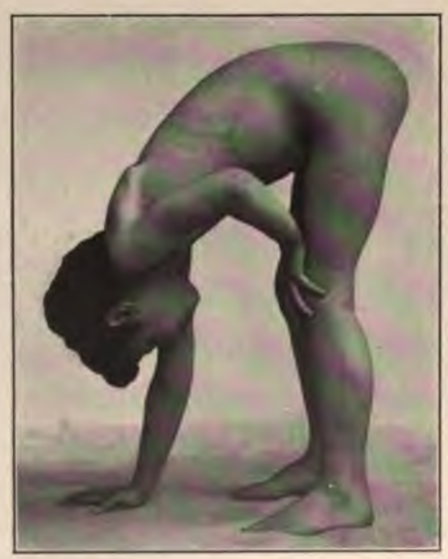
muscles of the thighs (Fig. 69), and an atrophy of the glutei muscles. An atrophy of the muscles of the back produces a condition

FIG. 72.



Pseudohypertrophic paralysis. The act of rising.

FIG. 73.

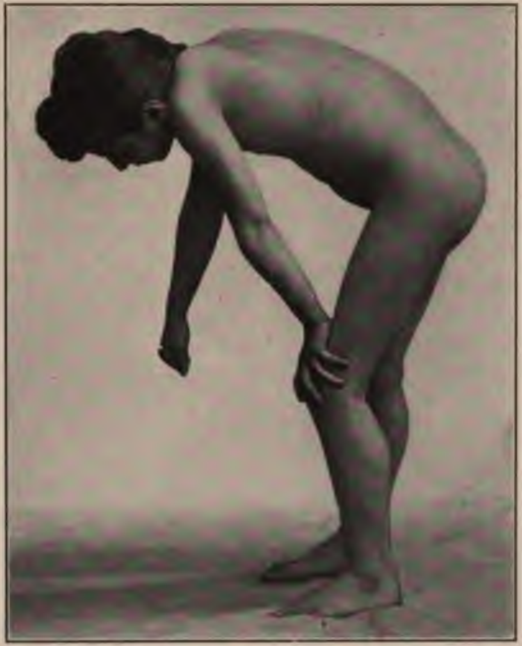


Pseudohypertrophic paralysis. The act of rising.

of forward curvature of the spine and causes the child to throw his shoulders back in order to preserve his centre of gravity. Even in the early stage of the disease, before the muscles about the scapulæ are affected, it is found that if a plumb-line be dropped from the scapula it will clear the buttocks. This is not possible in a state of health.

As the disease advances upward the arms become affected, the infraspinati are the first to be hypertrophied, so that the child appears to have a very prominent pad on the back of the shoulder-blades. The supraspinatus and deltoid may become hypertrophied also, and sometimes the triceps and biceps as well, but in the majority of cases these muscles atrophy without any deposit of fat. While these muscles hypertrophy other muscles about the shoulder become atrophied; thus the rhomboids and levator anguli scapulæ, and also the serrati become atrophied and paralyzed, and hence the shoulder-blades stick out from the back like a pair of wings. (Fig. 75.) Biceps and triceps may then in turn atrophy, rendering all motions at the shoulder-joint imperfect and making flexion and extension of the elbow very weak. As a rule, the muscles of the forearms and of the hands are not affected.

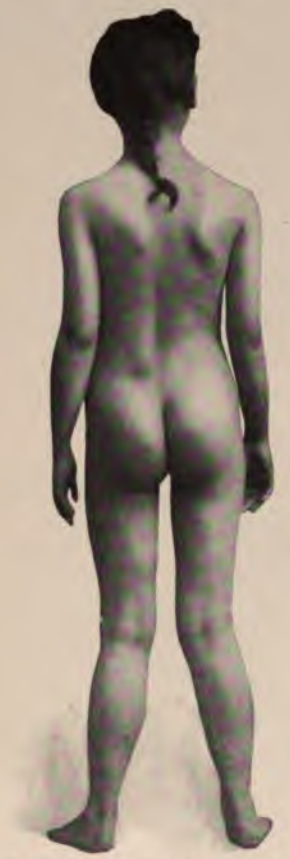
FIG. 74.



Pseudohypertrophic paralysis. The act of rising.

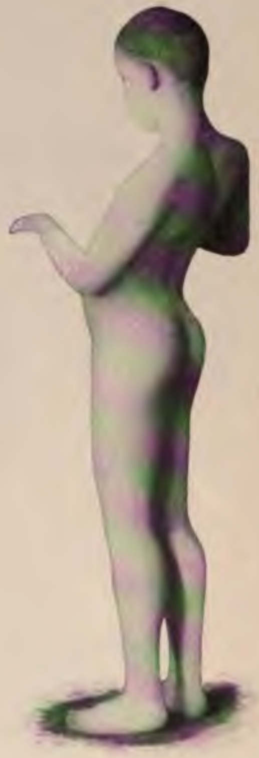
After the muscles are considerably atrophied, with or without the production of pseudohypertrophy, a contraction occurs, causing deformities in the joints. Thus talipes equinus is first produced, then a permanent flexion of the legs upon the thighs and the thighs upon the pelvis. Curvature of the spine, which can no longer be corrected by suspension, develops and often leads to lateral curvature by the unequal contraction of the muscles of the back upon the two sides. Contractions may occur also in the arms, producing a flexion of the elbow and interfering with passive movements of the shoulder-joint.

FIG. 75.



Pseudohypertrophic paralysis. The calves are large; the back is weak and curved forward. Deltoids and triceps are atrophied. Serrati are weak, hence the scapulæ protrude.

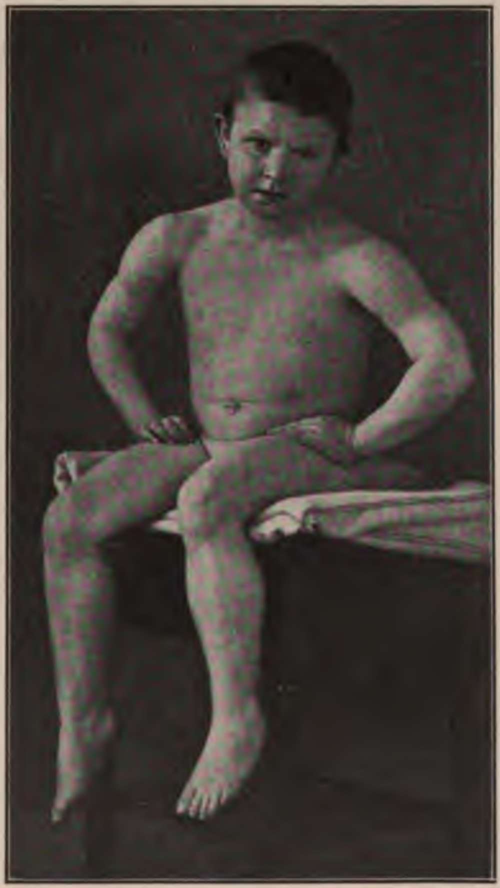
FIG. 76.



Pseudohypertrophic paralysis. The lordosis is well marked, and the attempt to preserve the balance by throwing the shoulders back is demonstrated.

The electrical contractility of the muscles is gradually reduced as the muscles atrophy, but there are no changes of the nature of reaction of degeneration. There are no fibrillary contractions in the muscles. Sensation is not at all impaired. The reflexes remain normal until the muscle becomes too much atrophied to respond. These facts enable a differential diagnosis from anterior poliomyelitis of the acute or chronic type to be easily reached.

FIG. 77.

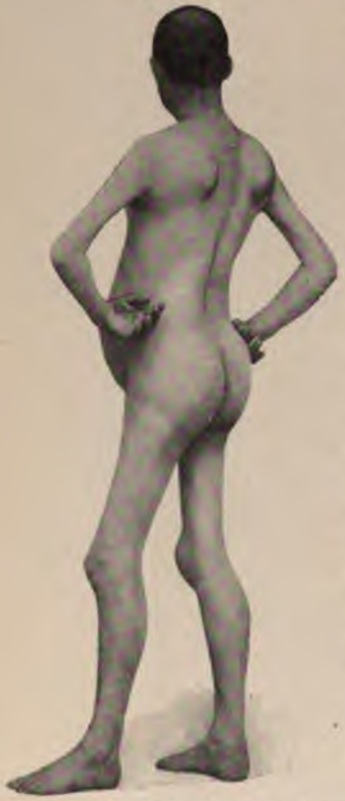


Pseudohypertrophic paralysis; five years after the onset. Muscles of arms and legs greatly hypertrophied. Both feet contractured and in a position of talipes. (Curschmann, Klin. Abbildungen.)

The course of the disease is slow and progressive through four to ten years, by the end of which time the child is rendered helpless, cannot stand or sit up in bed, or feed itself. The disease is not a fatal one, but in the enfeebled invalid state intercurrent diseases, especially of the respiratory organs, are the usual cause of death.

2. **Erb's Juvenile Form.** This type begins, as a rule, between the ages of twelve and sixteen; but cases have been observed which did not develop until after the age of twenty. In this form of muscular dystrophy the muscles of the shoulder are first affected, the pectorals, trapezii, latissimus dorsi, rhomboids and deltoid muscles become gradually hypertrophied, but at the same time are weakened. Peculiar deformities of the chest often develop, the shoulders being thrown forward and the scapulæ protruding. As the disease progresses the biceps and triceps become atrophied, but the muscles of the forearms and hands remain in their normal condition. Later on, if the disease does not come to a standstill, the muscles of the back become atrophied, lordosis appears, with deformities of the spine and difficulty in walking. Later the glutei and anterior muscles of the thigh are affected, either atrophied or hypertrophied, and finally the calf muscles and peronei become implicated, causing total paralysis with talipes equinus. In the majority of cases hypertrophy is more marked in the deltoid, infraspinatus, sartorius, and gastrocnemii. It will be seen that in the later stage of the disease the terminal condition is similar to that occurring in pseudohypertrophy, a fact which makes it evident that the only difference between these two types of dystrophy is in the direction of progress in the muscles involved.

FIG. 78.



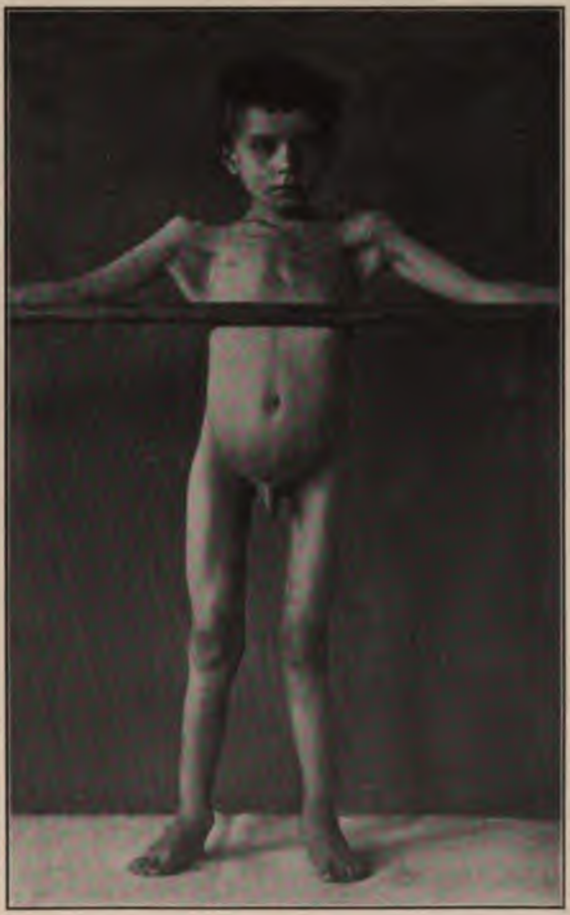
Erb's juvenile dystrophy. The disease began at the age of sixteen; photograph made at age of thirty. Supra- and infraspinati, and gastrocnemii hypertrophied. Trapezii, deltoids, biceps, triceps, latissimi dorsi, serrati magni, erectores spinæ, all muscles of thighs, and glutei are atrophied. (By permission of Dr. G. L. Walton.)

3. **Landouzy-Dejerine Type.** In this type, which develops usually in early childhood, but occasionally in adults, the feature is the appearance of the muscular atrophy in the face. It is true that both in pseudomuscular hypertrophy and in Erb's juvenile dystrophy the disease may eventually extend to the face, producing all the symptoms seen in the Landouzy-Dejerine type. The atrophy begins in the orbicularis oris and extends to the risorii, to the levator menti, and to the finer muscles of the face about the mouth. The result is that the lips become weak, cannot be firmly closed, and the mouth is habitually open and the lips are slightly

everted, producing a peculiar facial appearance that has been termed the "tapir mouth."

This form of paralysis affects the act of speech, linguals and labials being defectively pronounced, the ability to whistle, or to purse the lips, or to apply the lips firmly to a glass in drinking being lost. Frequently saliva runs from the patient's mouth, as in facial palsy. The affection is bilateral. The muscles about the eyes, as a rule, escape, so that winking is not interfered with, and the muscles of mastication and of deglutition are not involved. As time goes on in these cases the muscles of the shoulders and body, and finally

FIG. 79.



Progressive muscular dystrophy, atrophic type of Landouzy-Dejerine. Age, nine years; duration, seven years. Face, arms, and legs extremely atrophied. Face expressionless; pectorals and deltoids wholly atrophic; thighs atrophied. The forearms and the legs are normal. (Curschmann.)

of the legs, become involved, and the terminal stage of the disease is not unlike that in the two types already described. Fibrillary twitchings are not present, the electrical reactions are normal, but are progressively lost. There is no disturbance of sensibility. Mechanical excitability of the muscles is gradually lost as the muscles atrophy.

While these three types of muscular dystrophy are sufficiently different from one another to be easily recognized, it must be admitted that many patients suffering from the disease present symptoms common to two or more types. Even Erb admits the occurrence of transitional forms of dystrophy, and shows that the course of the disease may present variations in various members of the same family who are affected. In the terminal stage it is not always easy to ascertain to which type a patient is to be assigned.

The diagnosis of this affection from bulbar palsy is not difficult, because in dystrophy the tongue and muscles of swallowing are not involved.

Prognosis. The prognosis in muscular dystrophy is unfavorable. In all forms of the disease there is a tendency to slow progress, to an extension of the affection from muscle to muscle until finally a state of helpless paralysis with extreme emaciation is reached. This, however, is not an invariable occurrence, as I have seen several patients in whom the disease has come to a standstill and who have remained in a stationary condition for six or eight years without being incapacitated from walking. The prognosis as far as life is concerned is good, provided complicating diseases, especially of the respiratory organs, are prevented. These patients are peculiarly liable to develop tuberculosis of the lungs or pneumonia or bronchitis from disability in respiratory movements. Occasionally the diaphragm is involved and the patients die of respiratory paralysis.

Treatment. Treatment of muscular dystrophy must be by general measures to support the health and nutrition of the individual. A country life in the open air, with good food and moderate exercise, is advisable, and the systematic use of massage and gymnastic exercises, not carried to the point of fatigue, will often tend to reduce the rate of progress in the muscular dystrophy and in some cases to cause an arrest of its progress. Thus the patients alluded to in whom the disease has come to a standstill have had most skilful massage for years, daily for about one hour. Electricity has been used for the purpose of exercising the atrophied muscles, and when massage is impossible it may give good results. When the contractures have occurred the question may arise of the propriety of tenotomy, but in several patients in whom I have had it done it has not afforded any permanent relief, but, in fact, has been attended by an increase of the disability. The application of corsets or plaster-of-Paris jackets to correct the deformity in the spine is not to be advised, because the lordosis assists the individual in preserving his equilibrium, and if the spine is held erect in its natural posture the child falls forward and is unable to walk. Individuals suffering from muscular dystrophies are usually such chronic invalids as not to consider the question of marriage, but should this question arise it should be strongly advised against, inasmuch as this disease is so peculiarly hereditary.

MUSCULAR ATROPHY OF THE PERONEAL TYPE.

Charcot-Marie-Tooth Form of Progressive Muscular Atrophy.
Progressive Neural Muscular Atrophy of Hoffman.

In 1886 Charcot and Marie[1] in Paris, and Tooth[2] in Cambridge, England, described independently a form of progressive muscular atrophy beginning in the muscles supplied by the peroneal nerves and advancing upward as high as the knees in the legs, and subsequently attacking the muscles of the hands and forearms. This particular type of muscular atrophy has been recognized by many authors, especially by J. Hoffman[3] and B. Sachs.[4] Although it is an extremely rare form of disease, a sufficient number of cases can now be collected to establish the affection as independent both of the muscular dystrophies on the one hand and of anterior poliomyelitis on the other.

Etiology. In some cases there appears to be an hereditary disposition to the disease. Several members of a family may be affected, and in a few families it has been traceable through two or three generations. In many patients, however, no history of inheritance can be obtained. No etiological factors have been established, and syphilis does not appear to be a cause. In a few cases it has been preceded by some form of infectious disease. The disease uniformly appears in young persons before the age of twenty years.

Pathology. The cases first recorded were supposed to be of the nature of muscular dystrophy, a progressive atrophy of the muscles of the legs and forearms and of the feet and hands being the only lesion noticed. But in 1889 Hoffman discovered an atrophic neuritis in the peroneal nerves in addition to the muscular atrophy, and from that date the disease has been ascribed to a degenerative neuritis. Bernhardt,[5] in 1893, recorded a case in which in addition to the atrophy of the peroneal nerves there were found some changes of a sclerotic character in the posterior columns of the spinal cord; and Siemerling,[6] in 1899, after a study of all the cases upon record, reached the conclusion that the lesion of the disease consists in a degeneration both of the muscles, nerves, and posterior columns of the spinal cord, a lesion which involves the entire sensory element both in its peripheral and central prolongations as well as the motor fibres of the nerves. He has found the posterior spinal ganglion affected, and he has also found a sclerosis in a portion of the lateral columns of the cord not involving the pyramidal tracts. Siemerling is inclined to abandon the view that the disease begins in the peripheral nerves, and is inclined to ascribe it to a central lesion in the posterior horns of the spinal cord, but the exact pathology is still a matter of uncertainty.

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[1] Rev. de Méd., 1886.

[2] Brain, vol. x. p. 243.

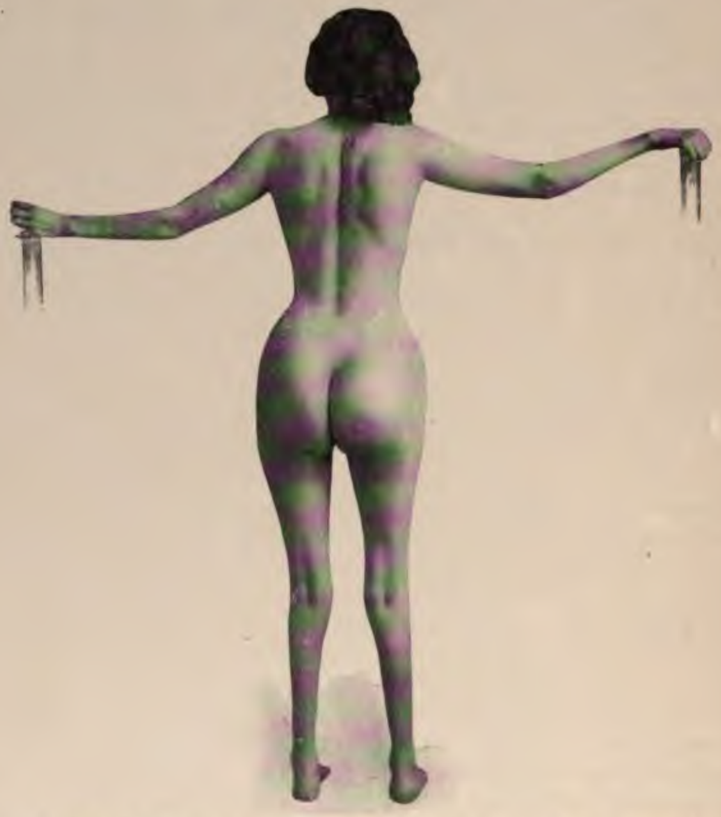
[3] Arch. f. Psych., Bd. xx., S. 560, and Deut. Zeitschr. f. Nerven., Bd. i., S. 95.

[4] New York Medical Journal, December, 1888. Brain, 1890.

[5] Virchow's Archiv, Bd. cxxxiii., S. 259.

[6] Arch. f. Psych., Bd. xxxi., S. 105.

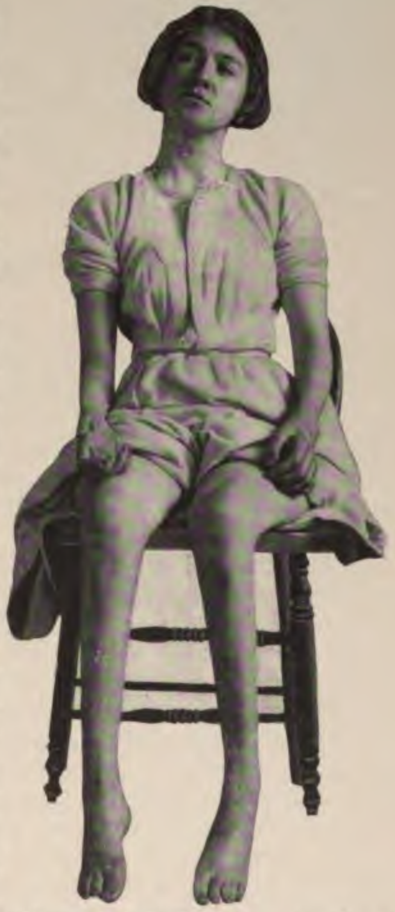
FIG. 80.



Charcot-Marie-Tooth disease. Atrophy of the legs below the knees and of the arms below the elbows.

Symptoms. The disease begins with a gradually increasing atrophy and weakness of the intrinsic muscles of the feet and of the long peroneal muscles on the outer side of the legs. The weakness and atrophy then appear in the anterior tibial muscles and extensor communis digitorum and in the posterior tibial muscles, so that after a slow increase during two or three years the patient is almost incapacitated from walking. The foot falls in walking, and hence the gait resembles the stepping gait of multiple neuritis. Occasionally the vastus internus in the thigh is atrophied and adduction of the knee becomes somewhat difficult. The affection of this muscle somewhat impairs the gait, allowing the knee to fall outward; hence the patients place the feet somewhat too far from one another in walking. (See Fig. 80.) The muscles which are affected show fibrillary contractions, they lose their reflex activity and mechanical excitability, and there is a progressive diminution of the electrical excitability for both currents. In many cases a complete reaction of degeneration appears in an early stage. As a rule the weakness and atrophy do not extend above the knees, and hence the contrast in the appearance of the thigh and leg is very marked in these patients. In all cases club-feet finally develop, and bilateral club-feet should always suggest this disease. The symptoms appear in the arms after the legs have been affected for some months or years. Occasionally, however, the hands and arms are affected from the beginning. There is a gradual atrophy and weakness of the intrinsic muscles of the hands, of the thenar and hypothenar groups, and also of the forearms, and as these become weaker abnormal positions of the fingers are assumed, giving rise to deformities of the nature of claw-hand. The muscles both on the front and back of the forearm are affected, and fibrillary contractures and diminution of electrical contractility with reaction of degeneration are found. The disease may come to a standstill at this point, as in the patient whose photograph is shown in Fig. 80, and there may appear to be no progressive increase in the symptoms for a long time. In other cases, however, the disease makes more rapid progress, the atrophy and paralysis involving the thighs and the arms, the muscles of the body and neck, and even the face, and the patient is gradually reduced to a skeleton, and dies from exhaustion.

FIG. 81.



Charcot-Marie-Tooth disease. Atrophy of the legs and drop-feet, and atrophy of the hands.

There are a few sensory disturbances. In some cases a slight numbness with diminution in the pain sense along the outer side of the legs is noticed, and in other cases complete anæsthesia in the distribution of the peroneal nerves has been found. The paralyzed limbs are usually cold and have a tendency to cyanosis, especially in cold weather.

The duration of the disease is uncertain, for many cases appear to be arrested, and the patients live their lives with a condition of atrophy in arms and legs and do not die of the affection. In other cases where the progress of the atrophy is more rapid they die of some intercurrent disease.

Prognosis for recovery is always unfavorable.

Treatment. A general course of tonic treatment with baths, massage, and electricity to the atrophied muscles appears to have some effect in lessening the rapidity of the atrophy, but does not appear to arrest the progress of the affection.

MYOTONIA CONGENITA. THOMSEN'S DISEASE.

This disease, described in 1876 by Thomsen,[1] who was himself affected, should hardly be included in a treatise on nervous diseases, but may be mentioned, inasmuch as it produces a certain amount of paralysis. It is a congenital and inherited disease of the muscles, characterized by a marked hypertrophy of muscular fibres and a proliferation of the muscular nuclei. The muscular fibres differ in shape from the normal, being more circular and not polygonal in transverse section.[2]

Symptoms. The symptoms of the affection are a peculiar rigidity of the muscles occurring after they have been at rest for some time. The muscle is so firmly contractured that the patient cannot overcome the tonic spasm, and hence moves with great difficulty until the spasm relaxes. All the muscles of the body may be affected, and hence walking, running, and the use of the hands are very much hampered. The quick compensatory and automatic movements necessary in order to preserve the balance are so markedly interfered with that these patients constantly lose their equilibrium and are liable to falls. By repeated attempts at movement and by constant exercise these muscles may be limbered up, so that at the end of a long walk these patients may walk in a normal manner, but any period of rest or the ordinary rest of night is always followed by a renewal of the hard contraction of the muscles. Patients are hampered all their lives by this abnormal rigidity. Cold or damp weather, cold baths, great mental excitement, and sudden sensory impressions are said to increase the degree of the spasm. Active exercises decrease it. The patients have no other symptoms whatever, and hence the disease cannot be mistaken for anything else.

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[1] Archiv f. Psych., Bd. vi., p. 702; also Bd. xxiv., S. 918.

[2] Jacoby. Journal of Nervous and Mental Disease, March, 1887.

The electrical contractility in the muscles is distinctly altered, a tonic contraction being produced by faradic excitement, and the reaction of degeneration being discovered on galvanic examination. Erb describes undulating rhythmical contractions starting from the cathode and passing to the anode. Mechanical excitability is increased, percussion of the muscle producing a sharp contraction of the muscle which remains for some time (fifteen to twenty seconds), producing a groove upon the skin. Pressure upon the muscle will also produce a firm contraction. As but thirty cases have been recorded in literature up to the present time, the affection may be considered rather a curiosity. There is no known treatment.

CHAPTER XV.

SYRINGOMYELIA OR GLIOSIS SPINALIS.

History. Syringomyelia (σῦριγξ, tube, hollow; μυελός, marrow) or gliosis spinalis is a disease of the spinal cord characterized by the production of a cavity within the cord of varying length. The name was given by Ollivier in 1824, but the condition was first described by Etienne in 1546 and is mentioned by numerous writers on anatomy from that time onward. Portal (1804) was probably the first to ascribe a form of spinal paralysis to this lesion on the basis of four cases which he observed. From his time, however, until 1860 the condition, though occasionally noted by pathologists, excited no interest. Then with the beginning of pathological study of the nervous system various hypotheses were proposed to explain the existence of cavities within the cord, and Lockhart Clarke, Vulpian, Hallopeau, Charcot and Joffroy, Leyden, Schultze,[1] and Kahler[2] made important contributions to the subject. In the monographs of Roth, Wichmann, and Anna Baumler (1889) over 100 cases with autopsies were collected and analyzed. The study of these cases from a pathological standpoint was soon followed by their analysis from the clinical side, and in 1887 Schultze[3] and Kahler[4] established the possibility of diagnosticating this condition during life. Their statements have been confirmed by clinical observers all over the world. In a monograph by Schlesinger[5] 526 references to published cases or discussions of the subject are given. Dimitroff[6] has recently added to this literature.

Pathology. The post-mortem appearances in a case of syringomyelia are very characteristic. The spinal meninges are normal. The contour of the cord is sometimes irregular, owing to a bulging at some places or a retraction at other places, or it appears at places flattened; sometimes it is not altered. Fluctuation may be detected by palpation. Usually a rupture occurs in the process of removal of the cord, and the fluid, a clear serum, runs out, leaving the cord partly collapsed. It is then evident that there is a long cavity within the cord, usually near the central canal, but sometimes so extensive as in a cross-section to leave merely a thin ring or wall of cord tissue. Sections of the cord at various levels will

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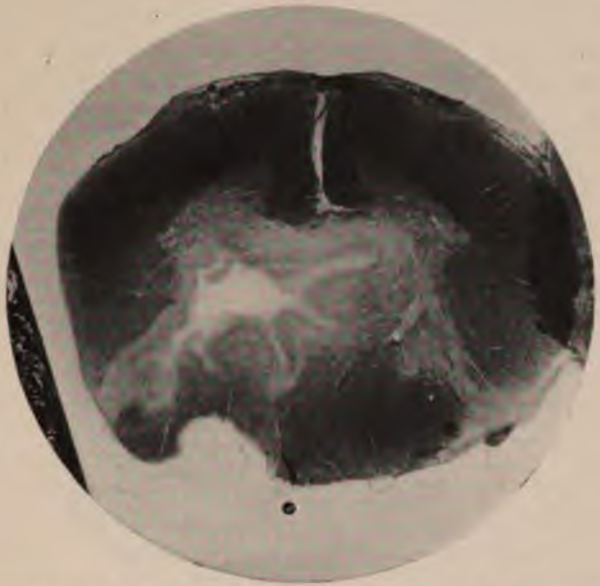
[1] Schultze. Virchow's Archiv, Bd. lxxxvii. and civ.
[2] Kahler. Vierteljahrschrift für Pract. Heilk., 1879. Arch. f. Psych., Bd. x.
[3] Schultze. Zeitschr. für klin. Med., Bd. xiii., 1887.
[4] Kahler. Prager med. Woch., 1888, Nos. 6 and 7.
[5] Schlesinger. Die Syringomyelie, 1895, Leipzig.
[6] Arch. f. Psych., Bd. xxxv. S. 42.

demonstrate that this cavity extends for some distance through the cord, and that it varies in size and shape at different levels. The usual situation of the cavity is in the lower cervical and dorsal regions. In some cases it is short, not involving more than five or six segments; in others it is long, extending through the entire length of the cord and upward into the medulla and pons. Sometimes two or three separate cavities have been found at different levels. All possible variations have been observed in different cases. In some cases a tumor has been found on one side or within the wall of the cavity.

When the cord is hardened, cut, stained, and examined microscopically it presents certain characteristic appearances. The cavity may be of any size or of any shape, but lies chiefly near the central canal, behind the anterior commissure, or in the posterior central gray matter, or in a posterior horn, or in both horns of the cord. In some cases it invades the central gray matter and the anterior horn or horns, but it is rarely symmetrical in its invasion of the cord tissue on the two sides. In some cases the gray matter is entirely replaced by the cavity. In other cases the cavity has invaded the white columns of one or both sides. The posterior columns are more frequently invaded than the lateral or anterior. In the most extreme cases it appears as if all the cord tissue had been destroyed. The cavity is then surrounded by a thin wall forming its sac, and no trace

FIG. 82.

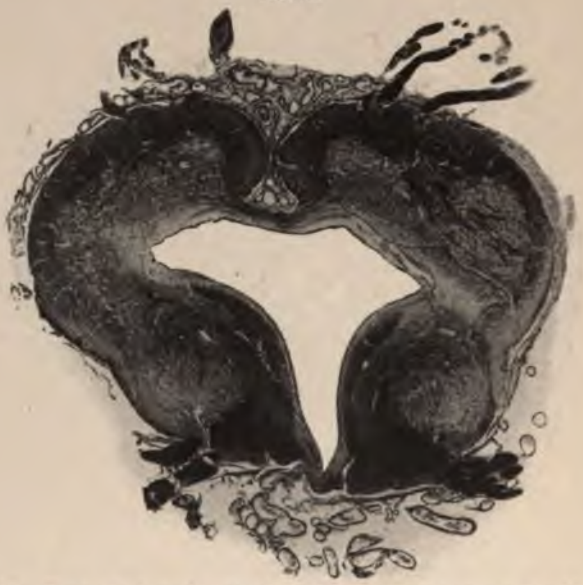


Syringomyelia. Cavity in the central gray matter of the left half, with numerous diverticula. Walls of cavity formed by gliomatous tissue infiltrating the spinal cord. Weigert stain.

of gray or white substance remains. The wall of the cavity is smooth, but here and there papillary projections occur upon it.

The cavity is usually surrounded by a zone of thick neuroglia tissue which stains deeply with carmine and hematoxylin, is unstained in the Weigert hematoxylin stain, is deeply stained by the Weigert neuroglia stain, and by the Golgi stains. The thickness of this neuroglia wall varies in different cases. Its structure is most dense near the cavity; but it is thinner in the adjacent parts and fades away into the normal cord, not having as a rule a sharp boundary. (See Fig. 82.) Under a high power of the microscope it is seen to be made up of fine fibres, of nuclei, and of small and large neuroglia cells, a few of which are seen in some cases to be in a state of vacuolization and progressive liquefaction and to be breaking down. A fine filamentous network, containing spider cells and spindle-shaped cells with long processes and many nuclei, extends outward into the nerve tissue—the appearance being that of a partial infiltration of the normal cord by gliomatous elements, the degree of which is greatest near to the wall of the cavity. Such an infiltration of the cord with cells is also found in the segments above and below the limits of the cavity, especially about the central canal. The cavity frequently occupies the place usually taken by the central canal. Sometimes it is seen to communicate with the remains of the central canal, and then some epithelial elements may be found in its wall.

FIG. 83.



Syringomyelia. The cavity has taken the place of the central gray matter, and has invaded both posterior columns.

In other cases the canal is pushed to one side and lies in the wall of the cavity. In a few sections there may appear to be two cavities side by side; but a careful examination of sections above or below will show that one of these is really a diverticulum from the main cavity. The cavity itself is never entirely lined by cylindrical epithelium; but in a few cases one side of it may be so lined, and in these there is a manifest absorption of the original central canal into the new cavity, with more or less proliferation of the lining epithelium. A few cases have been described in which a true glioma or sarcoma filled the cavity, being an evident outgrowth from its wall.[1] Changes in the bloodvessels of the cord are sometimes observed. There are very few capillaries to be seen in the wall of the cavity; but outside of it, in the adjacent parts of the cord where the infiltration of small cells and nuclei is seen vessels are more numerous than normal, their calibre is larger, and they are more tortuous than usual. In some cases distinct thickening of their walls has been noticed. Capillary hemorrhages are often found within the gliomatous structure.

Various hypotheses have been proposed to explain the conditions described. They may be briefly summarized as follows:

1. It has been supposed that syringomyelia always originates in a congenital defect in the development of the spinal cord. It has been thought that the central canal of the cord may be unduly distended during fœtal life and early infancy by fluid, thus remaining as an unusually large cavity within the cord, around which cavity there subsequently develops a proliferation of the embryonal epiblastic elements or a thickening of the normal glia tissue, which is known to be more abundant in this situation than elsewhere in the cord. Such a cavity is lined by epithelium and is described by many pathologists under the name "hydromyelus." But some believe that hydromyelus may pass into syringomyelia by the proliferation of glia cells, the invasion of the normal tissue, and its subsequent breaking down. They thus explain the admitted fact that the degree in which the cavity in syringomyelia is lined by cylindrical epithelium varies. Others hold that in the closure of the central canal during embryonal development a portion of it is shut off from the main canal so as to leave a subsidiary canal in the posterior septum of the cord, lined with epithelium and surrounded, as is the normal central canal, by epiblastic tissue which subsequently proliferates, constituting a neuroglia hyperplasia, and then breaking down. Those who hold this opinion, therefore, ascribe all cases of syringomyelia to a congenital malformation of the cord and believe that the cavity of syringomyelia has a necessary relation to the normal central canal and usually communicates with it.

2. Another view of the disease is that the normal glia structure of the spinal cord, which is thickest about the central canal, undergoes

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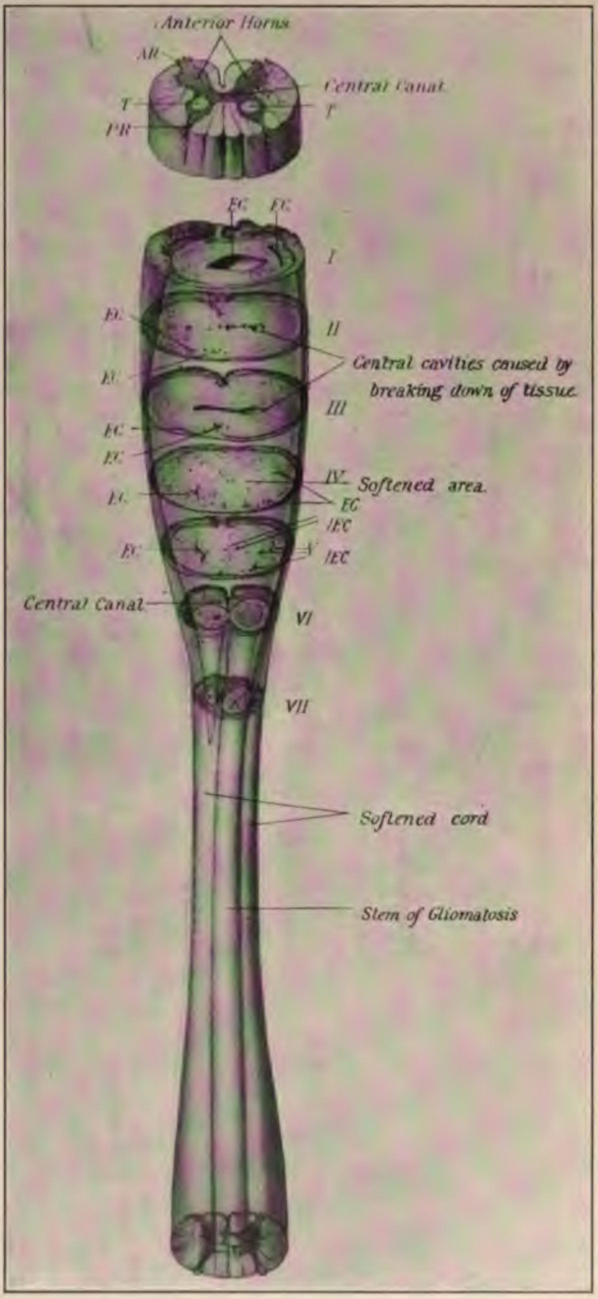
[1] Van Gieson. Journal of Nervous and Mental Disease, July, 1889.

FIG. 84.



Glioma of the spinal cord, with formation or cavities within it. *A R*, anterior nerve roots *T*, tumor; *P R*, posterior nerve roots; *E C*, epithelial lined cavities within the tumor. (Hudson, American Journal of the Medical Sciences, cxvii., 648.)

a proliferation from some unknown cause—possibly an irritant poison in the fluid of the central canal;[1] that this gliomatous new structure extends outward into the adjacent tissue, both into the gray and white matter, and subsequently breaks down in its centre, the cells becoming liquefied and disintegrated; thus a cavity originates within a gliomatous mass. This cavity at its origin has no necessary relation to the central canal; but inasmuch as the gliomatous proliferation begins, as a rule, near to the canal, the cavity usually breaks into the central canal and thus makes a communication with it. The wall of the cavity is described as consisting of hyperplastic neuroglia with larger and smaller branching neuroglia cells, and small spheroidal cells, and oval cells lying in a network of fibres, at places closely packed together, at places loosely arranged, so that it has a porous meshwork structure. This forms a sort of limiting membrane for the cavity, but beyond it within the nervous tissue there is an infiltration of glia cells. These cells are seen to be in a state of liquefactive degeneration both in the wall of the cavity and elsewhere. In some cells the nucleus is attached to or surrounded by the homogeneous remains of the cell body. Some cells are converted into sacs of fluid. Thus there is a manifest tendency in the glia cells to break down, and the cavity is the result of such disintegration. Those who hold this opinion have named the disease spinal gliosis, believing the glia proliferation to be the essential factor in the pathology. Some consider this an inflammatory process;[2] others[3] deny anything more than a simple hyperplasia.

Investigations of Weigert[4] upon the structure of neuroglia seem to establish that new formations of glia may be either cellular in structure or fibrous in structure. If cellular, the new formation is a true glioma, such as is found in tumors of the brain or spinal cord, and in such a glioma fibres are few. Weigert calls attention to the fact that this is not the structure of the glia tissue about the cavity of syringomyelia, but that the neuroglia found around this cavity consists almost exclusively of glia fibres with few cells; and that these fibres, though extending in all directions, are chiefly vertical in their course. Miura also has shown the sharp contrast between ordinary glioma, even glioma containing a cavity, and the gliomatous condition of the cord in syringomyelia. Weigert holds that the neuroglia is merely a substance produced by nature to take the place of nerve tissue which has been destroyed, and that its proliferation is always a sign that the nerve tissue has primarily disintegrated. Such destruction of nerve tissue would, therefore, according to his view, precede the formation of gliomatous tissue; hence he wholly discards the hypothesis of syringomyelia to which the name spinal gliosis has been applied. Weigert says: "Many authors believe that the essential lesion in syringomyelia is the formation of a tumor followed by softening and the formation of a cavity. It is admitted that there is a growth of neuroglia of the typical fibre type about the cavity. But this fibre mass, devoid of cells, does not resemble a glioma and there is no reason to believe from the mere presence of neuroglia that the cavity is not a congenital or acquired abnormality of the central canal. There is a thick cluster of neuroglia fibres normally about the canal. By the pressure in this enlarged canal the nervous tissue may be destroyed, and hence a growth of neuroglia fostered. If the pressure increases the neuroglia may also be destroyed, and in its place about the cavity a hyaline formless mass may remain. The gliosis is not the essential feature, it is only a secondary result." Turner,[1] however, has described a case in which there is a gradual and direction transition between a true glioma and a gliomatous infiltration of the cord with the production of a cavity.

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[1] Babes and Manicatide. Archives des Sci. Méd. de Bucharest, May, 1896.

[2] Miura. Ziegler's Beiträge zur path. Anat., xi., 91.

[3] Schultze. Zeitschr. für klin. Med., xiii.

[4] Weigert. Beiträge zur Kenntniss der normalen menschlichen Neuroglia. Frankfort, 1895.

3. Many authors have observed, subsequently to disease of the spinal arteries, the formation of cavities in the cord independent in their situation of the central canal. Thus Müller and Medin have seen a cavity in the gray matter of the cord with walls of normal nerve tissue with no signs of inflammatory disease or of proliferation in the glia, the size of the cavity corresponding in situation to the degree of endarteritis in the spinal vessels and having no definite relation to the central canal. Wieting has described a cord containing numerous cavities due entirely to the low nutrition of the nerve tissues, and consequent necrosis from disease of the spinal arteries in connection with meningo-myelitis. It has been thought by Kronthal that lymph stasis within the cord, and consequent necrosis, produced by transverse compression of the cord, may lead to the formation of cavities which may or may not communicate with the central canal; but this surmise is doubtful, since compression by tumors or after Pott's disease is not found to cause cavities. The supposition that a cavity in the cord may be due to a diseased condition of the bloodvessels seems proven in some cases; but such cavities do not resemble those of syringomyelia, and the suggestion finds little support in Weigert's hypothesis regarding the function of neuroglia. For if this hypothesis be correct a neuroglia growth would occur to replace the disintegrated nerve tissue, and in the attempt of nature to fill up the empty space would be thickest about the cavity. Necrotic cavities, however, rarely have a well-marked wall.

4. Van Gieson has recently described a condition which he calls hemato-myelo-porus, of perforating hemorrhage in the cord, with the production of a long, narrow cavity. Such a cavity is occasionally surrounded by thickened glia tissue. Van Gieson shows that some cases which have been described as syringomyelia have really

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[1] Turner and Mackintosh. Brain, 1896, Pt. lxxv.

been old cases of hemorrhage. Turner and Mackintosh point out that the presence of a fibrin-like material, which they describe in some of the cavities in several cases of gliomatosis of the cord, suggests that hemorrhage assists in the formation of these spaces.

5. Lastly, there is not wanting an hypothesis which would trace it to bacterial infection. Prus,[1] from a careful study of Morvan's disease, reached the conclusion, which several authors had already announced, that Morvan's disease and syringomyelia are identical. He calls attention, however, to the fact that Zambaco maintains that it is identical with lepra anæsthetica, the bacillus of which can be recognized. And he appears to be willing to admit that the three diseases are, in fact, due to the same cause, namely, an infection of the nervous system by a germ which, in the lighter forms, attacks the peripheral nerves only and in the more severe forms attacks the spinal cord. In this view the neuroglia formation is set up by the irritation of the bacillus, and the cavity is due to the disintegration of the gliomatous substance. This hypothesis is strongly combated by Babes, who has observed six cases of lepra in which the bacilli were found in the cells of the cord, but in which there was no lesion resembling that of syringomyelia.

It seems evident, therefore, from a review of these various hypotheses, that cavities may be formed within the spinal cord under varying circumstances and by various pathological processes. First, from congenital defects of development; secondly, by a disintegration subsequent to a neuroglia proliferation either of inflammatory origin or of spontaneous occurrence; thirdly, as the result of retrograde metamorphosis of tissue, the nutrition of which is impaired by obstruction to the circulation; fourthly, by actual destruction of the cord by hemorrhage.

The time has not yet come to establish conclusively any one of these views of the origin of syringomyelia. In fact, until cases are observed at the outset of the disease, as well as after a long duration, no basis for a conclusion can be established.

When the cavity has existed for some time it is not uncommon to find evidences of ascending and descending degeneration in the columns of the cord, which are secondary to pressure or to the destruction of tissue at its point of maximum extent. Degeneration in the motor nerves and atrophy of the muscle fibres are also parts of the lesion in this disease. The various trophic disturbances in the bones and skin also require mention.

Symptoms. The diagnosis of syringomyelia rests upon the presence of three characteristic symptoms which, in the majority of cases, are present together. The existence of one of these symptoms alone should excite suspicion of the possibility of the disease being present. The presence of any two of them make the diagnosis very probable. These symptoms are, first, a loss of the sensations of pain and of temperature in any part of the body, tactile sense being preserved in the analgesic area; secondly, trophic disturbances in the skin, muscles, bones, or joints; thirdly, progressive muscular atrophy attended by paralysis.

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[1] Arch. f. Psych., Bd. xxvii., S. 771.

In addition to these symptoms there may be (*a*) a spastic paraplegia, or (*b*) disturbance of tactile sense with pain, or (*c*) the general symptoms of transverse myelitis in case the disease invade respectively the (α) lateral, or (β) posterior columns of the cord, or (γ) its entire area. Such an extension is not uncommon, and hence these symptoms must be considered as a frequent complication.

The distribution of the characteristic symptoms of the disease will depend entirely upon the extent of the lesion in the cord. As this lesion usually begins in the cervical segments the symptoms almost always appear in the hands. If the lesion be limited to one or two segments of the cord the symptoms will be very limited; but if it extend throughout the entire length of the cord and upward through the medulla and pons to the crus, the symptoms will be widespread and will involve the cranial nerves. The course of the disease is a very chronic one, the symptoms coming on slowly at any age, and often reaching a certain point and remaining stationary for years, the life of the patient being ended, as a rule, by some intercurrent disease, though occasionally sudden death is caused by the rupture of the cavity.

The disturbance of sensation, called by Charcot dissociated anæsthesia, is the chief characteristic of the disease. It is a symptom which is frequently unknown to the patient until it is demonstrated by the physician, although occasionally among the working classes, who are much exposed to injuries, the patient may have noticed that such injuries, especially burns, were not attended by pain. It is found upon examination of these persons that pricking, or cutting, or burning, or freezing of the affected area is not attended by sensations of pain, or of heat or cold, though the sense of touch is preserved. The sense of heat may be impaired when that of cold remains, or conversely. The sense of pain is a great protection to the body, giving warning of injury and assuring care and rest of the part; hence its absence exposes these patients to the risks of serious affections of the skin and joints, the consequence of neglect of small pathological processes at their start. The sense of touch is not often affected at all, although in cases where the cavity progresses to a considerable size and invades the posterior columns of the cord it may become somewhat blunted. The muscular sense appears to be preserved, excepting in this last class of cases. It is from these phenomena of dissociated anæsthesia that the conclusion has been reached that the paths of sensations of pain and temperature differ in their location from those of tactile sense, and that they pass into the central portion of the gray matter of the spinal cord soon after their entrance. It is certain that their entire course from below upward is not in the gray matter, otherwise a limited lesion of this portion in the cervical segments would produce a disturbance of these senses in the entire body below the lesion; but it appears that these sensations on their way from the surface of the body to the centripetal white columns of the cord (the antero-lateral tracts—see page 191) traverse the gray matter at the level at which they enter; hence the distribution of this disturbance of sensibility corresponds exactly to the position of the lesion in the spinal cord. As the exact area of the skin related to the individual segments of the cord has been determined, it is possible to reach a diagnosis of the exact extent of the spinal lesion by determining the exact extent of the analgesia.[1] The diagram on Plate XIII. shows this relation so far as it is at present determined. It demonstrates that the various districts of the skin can be assigned to the various segments of the cord. The distribution of the analgesia in syringomyelia is usually irregular, rarely symmetrical on the two sides. Inasmuch as the affection is more common in the cervical region the condition of analgesia is more frequently found in the hands and arms. It is found that small injuries to the fingers are not attended by pain, an abnormality which first directs the patient's attention to the existence of his disease. In the early stages of the disease there is merely a decided blunting of the sensations of pain and an inability to distinguish between slight variations of temperature, or certain sensations only are not perceived. Thus Dejerine records a case in which the thermal sense was lost for all temperatures above 68° F. The area of analgesia may not coincide exactly with that of loss of temperature sense. Sensations of cold or of burning, or sharp pains, sometimes precede the loss of sensation. When the disease is fully established the patient cannot distinguish any difference between iced water and boiling water, and a deep incision may be made without the slightest pain.

Trophic disturbances are a very frequent symptom in syringomyelia. In the majority of patients it is evident that the origin of these disturbances is some injury, wound, or burn that had not been observed on account of the loss of pain sense, and that had therefore been neglected, had become infected, and had gone on to ulceration or suppuration. In some cases, however, it is impossible to ascribe trophic disturbances to this cause, and the hypothesis of the existence of trophic centres in the spinal cord presiding over the general nutrition and the repair of the body receives its chief support from the facts observed in this disease.

The skin is the seat of the chief trophic disturbances. These may be of various kinds. There may be localized hyperæmia or anæmia of the skin. There may be changes in the perspiration, the part being abnormally covered with sweat or abnormally dry; and in addition to the acute inflammations of the skin already mentioned as produced by injuries, cases have been observed of serous exudation with desquamation, gangrene of the skin and subcutaneous tissue, bullæ and peculiar hypertrophies and atrophies of the skin.

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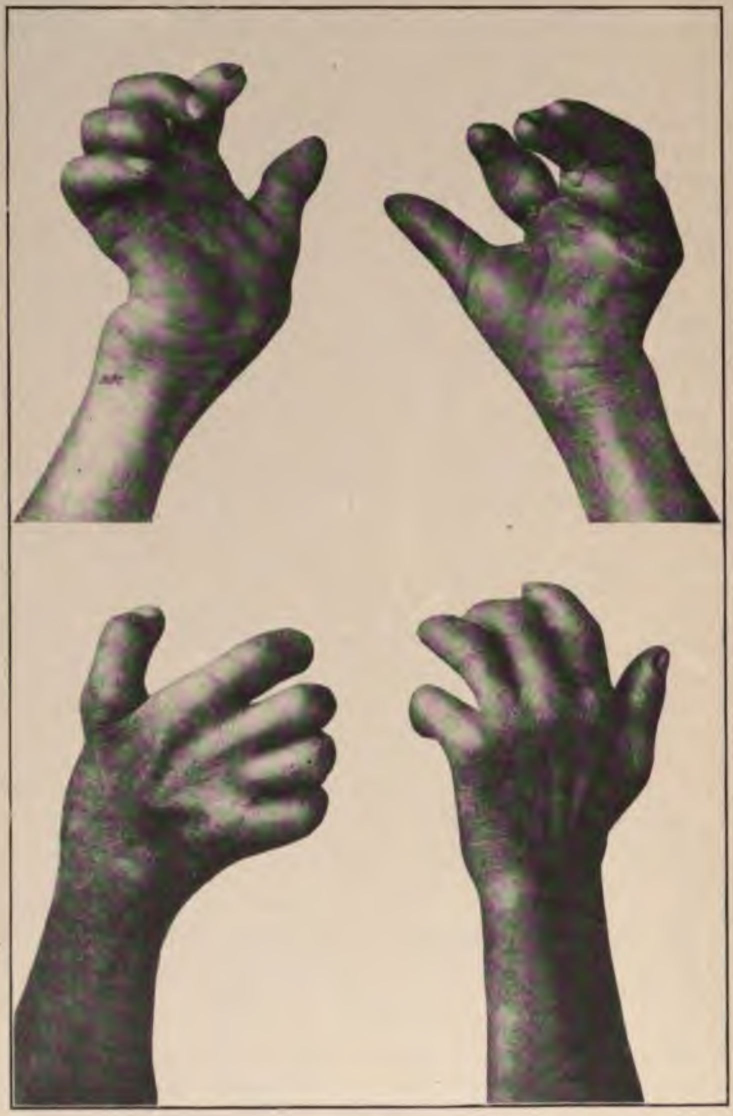
[1] Max Laehr. Arch. f. Psychiatrie, 1896, xxviii., 773.

FIG. 85.



Trophic changes in the hands in syringomyelia in the form known as Morvan's disease. The muscles are atrophied. The hands are much deformed. The skin is atrophied. The nails have fallen. The bones are hypertrophied at some parts, atrophied at others. The tip of one finger is eroded. (Curschmann, Klin. Abbildungen.)

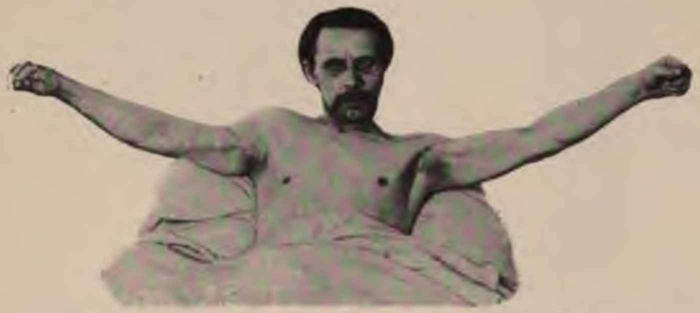
Another trophic disturbance which has excited much interest is the appearance of painless whitlows and small abscesses upon the fingers.

Morvan[1] described a disease occurring in a seaport of France among fishermen, in which felons appeared upon the fingers, producing deep ulcerations and even necrosis of the terminal phalanges. These were associated with other trophic disturbances of the skin and nails, and with analgesia. This disease, which was named after Morvan, is now thought to be a variety of syringomyelia, for in all cases examined after death a cavity has been found in the spinal cord. Fig. 85 shows the appearance of the hands in Morvan's disease.

The growth of the nails is commonly affected in the disease. They are hypertrophied, ridged, and occasionally stained. They become particularly brittle and are irregular in their form.

Affections of the joints and bones are very frequently observed in syringomyelia. In fact, there is no nervous disease in which joint affections occur so commonly as a complication. The shoulder, elbow, and wrist are the joints most commonly affected. In this respect the disease offers a contrast to tabes, in which the joint affections most frequently occur in the lower extremities. The character of the joint affection, is, as a rule, quite similar to that described by Charcot as occurring in locomotor ataxia—a large effusion within the joint, with great thickening of all the tissues, and later an absorption of the bones with an atrophy of the joint surfaces. Fig. 86 shows the appearance of a patient suffering from an arthropathy of the right shoulder, and Fig. 87 shows the condition found in the bone after death. Schlesinger has collected sixty-three cases of joint affection occurring in the course of the disease, and he estimates that this complication occurs in more than 10 per cent. of the cases. Alterations in the condition of the long bones are observed in syringomyelia, and spontaneous fractures, due to a spongy and brittle condition of the bones, have been recorded by a number of observers. Both the joint affections and these fractures proceed

FIG. 86.



Arthropathy of the right shoulder in syringomyelia. (Dercum.)

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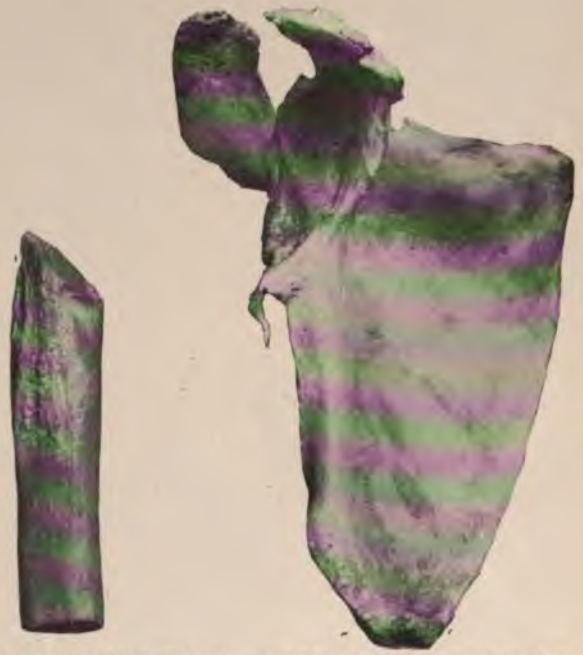
[1] Gazette heb. de Méd. et de Chir., 1883.

without pain to the patient, and hence are often neglected for some time after they begin.

In a considerable number of cases a marked curvature of the spine, either lateral or forward, and occasionally backward, has been observed. This has been ascribed by some authors to atrophy and weakness of the spinal muscles and by others to actual changes in the bones. Both conditions may occur. The spine is, as a rule, sensitive to pressure. Deformity is more likely to occur in the upper portion of the dorsal region than elsewhere. It is never very extensive.

FIG. 87.



Humerus and scapula of the right side. The head of the humerus has been destroyed. The glenoid surface of the scapula is enlarged, and its edges are thickened by bony deposits. The coracoid process presents peripheral accretions. (Dercum.)

As the records of the disease have increased its incidental association with various diseases—acromegalie, hysteria, paralysis agitans—has been recorded. Such associations have no particular significance.

Muscular atrophy attended by paralysis is present in more than one-half of the cases of syringomyelia. It usually begins as a progressive muscular atrophy invading the hands, especially the first lumbricalis muscle, then the thenar and hypothenar eminences, finally producing claw-hands (*main en griffe* of Duchenne), and then advancing up the limb to the forearm, arm, and shoulder. Occasionally the shoulder muscles are the first to be affected, and then the atrophy appears in the deltoid and scapular muscles, and later invades the biceps and supinator longus. The muscles of the spine are particularly liable to be invaded by the atrophy and paralysis, and as a consequence curvature of the spine is a very frequent symptom in this disease. The legs are less frequently affected (12 per cent. of the cases), but atrophic paralysis of the thighs and of the legs below the knee with consequent contractures has been seen.

The exact distribution of the atrophy and paralysis depends upon the extent of the lesion in the various segments of the cord. In the table already given on page 173 the relation between the various muscles of the body and the various segments of the cord is shown. It is therefore evident that from a knowledge of the muscles invaded a conclusion can be reached as to the extent of the lesion. The atrophic paralysis of the muscles is attended by fibrillary contractions and tremors and by a gradual diminution in the mechanical and electrical contractility of the muscle. It is only in the last stage of the disease, when the muscle is extremely atrophied, that it presents the reaction of degeneration.

The spinal reflexes may be disturbed in this disease. When the symptoms are located in the arms, elbow and wrist reflexes are lost, while the patella reflex is, as a rule, increased. If, however, the disease invades the lumbar region of the cord the patellar reflex may be lost on the side of the lesion.

In a few cases in which the sacral region of the cord has been diseased a loss of control of the bladder and rectum has occurred.

The spinal centre of the cervical sympathetic nerve lies in the first dorsal segment of the cord, and as this segment is very frequently affected, symptoms of paralysis of the sympathetic of one or both sides are commonly to be detected. They are a narrowing of the palpebral fissure, a retraction of the eyeball, sluggish pupillary action, with imperfect dilatation, a flattening of the side of the face, and a defective secretion of sweat.

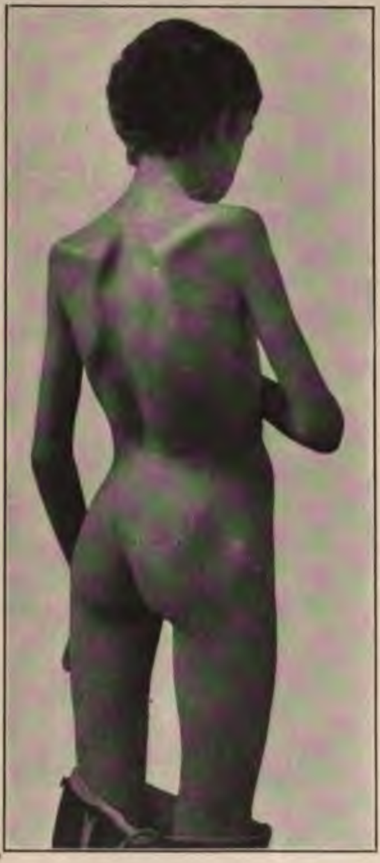
The extension of the disease to the medulla may cause symptoms referable to the implication of the cranial nerves. Atrophy with fibrillary tremor in the tongue and facial muscles, ocular palsies with nystagmus, and dissociated anæsthesia of the face and head have been observed. In a few cases paralysis of the vocal cords, disturbances in the act of swallowing, difficulty of respiration, and irregular heart action have indicated that the vagus centre has been affected. These symptoms are most serious, as sudden death commonly ensues.

The course of the disease is a chronic one. It advances slowly, and the symptoms are, as a rule, well established before the disease is recognized. The patients remain for months in a stationary condition or the paralysis slowly increases until they are disabled. The symptoms may finally extend to the entire body, although this is rare. Death occurs either from heart failure, or from cystitis, or bed-sores, or from some intercurrent affection, or rarely very suddenly without apparent cause, really from a rupture of the cord allowing an escape of fluid from the cavity.

The following history of a case of syringomyelia under my observation at the Vanderbilt clinic illustrates the usual symptoms and course of the disease:

P. S., of healthy parentage, noticed in March, 1896, when he was sixteen years of age, that he was becoming weak and clumsy in his hands, that he was dropping things unintentionally, and was losing strength in his arms. These symptoms were noticed in the left arm before they appeared in the right. They were not attended by pain or any noticeable sensory disturbance. It was noticed that his hands and arms became gradually thinner as they became weaker, and the emaciation soon extended to his body, especially about the muscles of the chest, and back, and scapulæ. He was not aware of any sensory disturbance until the time of his first examination at the clinic in January, 1897. He had had no trouble with his bladder or rectum. He had had no symptoms in his legs, excepting a slight weariness on any exertion, but he had noticed that his back had gradually become crooked, the right side of his body appearing to bulge. It was evident from the history that all his symptoms had made such gradual progress during the year that they had not attracted much attention until his inability compelled him to quit work.

FIG. 88.



Case of syringomyelia. Atrophy of the muscles of the shoulders and right arm. Curvature of the spine forward from atrophy of muscles of the back.

Examination in January, 1897, showed a very marked condition of atrophy with corresponding paralysis in the muscles of both upper extremities, chest, scapulæ, and back, as shown in the picture. (Fig. 88.) No muscle was entirely paralyzed, but all the muscles were extremely weak, presented fibrillary contractions on exposure to cold or on percussion, but did not show any reaction of degeneration. The atrophy was most extreme about the muscles of the scapulæ and in the deltoids and upper part of the arms. The muscles of the thorax and back were markedly atrophied, so that a lordosis was very evident, causing peculiar motions of balancing in the act of walking. The atrophy was about equal on both sides. The biceps was less atrophic than the other muscles of the upper arm. The flexors and extensors of wrist and fingers, the thenar and hypothenar muscles, and the interossei of the hand, were very much atrophied. The lower portion of the pectoralis major on both sides was preserved, but the upper part was atrophic. The muscles of the abdomen and legs were not in any way affected, but the knee-jerks were very much exaggerated, and there was ankle clonus on both sides. The elbow and wrist reflexes were lost. Face was normal. Sensation to touch was preserved in all parts of the extremities, body, and thorax, but sensations of heat and cold and of pain could not be perceived over the upper part of the thorax or in both upper extremities. The loss of pain sense was somewhat less extensive than the loss of sensation to heat and cold on the back. This patient was observed very carefully in St. Luke's Hospital for six months, but there was little or no change in his condition.[1]

Diagnosis. When the three characteristic symptoms already mentioned are present in any case there is no question regarding the diagnosis of syringomyelia. In the early stages of the disease, however, before all three symptoms appear, the disease may be mistaken for other spinal affections. Thus many cases are regarded for a considerable time as cases of progressive muscular atrophy or of chronic anterior poliomyelitis, and it is only on the appearance of the peculiar sensory disorder or of the trophic symptoms in the skin or bones that the first diagnosis becomes questionable. In other cases the early suspicion may be of tabes, especially if the disease be located in the lower part of the cord, for then the pains, especially the burning sensations, the disturbances in temperature and pain sense, and the paræsthesia, with trophic disturbances in the joints and loss of tendon reflex at the knee may suggest locomotor ataxia, even though the ataxia be not manifest. Then it is only when atrophies of the muscles and paralysis occur that syringomyelia is suspected. The diagnosis from amyotrophic lateral sclerosis may be made from the fact that in that disease there is an increase of mechanical excitability in the paralyzed muscles, an increase of reflex action, the early appearance of a spastic gait, an absence of sensory symptoms, and little tendency to trophic disorders.

A general myelitis or a disseminated myelitis may be diagnosticated in cases of syringomyelia when both motor and sensory and trophic disturbances are present; but the lack of symmetry of the sensory disorders and the peculiar preservation of tactile sense in syringomyelia should enable the observer to avoid this mistake. It is, however, to be remembered that in some cases the two diseases occur together.[2]

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[1] For other histories the reader is referred to the American Journal of the Medical Sciences, May, 1888, and December, 1896.

[2] Gowers. Diseases of the Nervous System, vol. i. p. 339.

While it is true that syringomyelia is often due to a tumor of the spinal cord, especially glioma, it is to be remembered that tumors of the cord are usually limited in extent to two or three segments, that they produce more widespread symptoms than syringomyelia, especially in the body below the level of the lesion; that the symptoms resemble those of a transverse myelitis of rapid onset, and that pain of a severe character is a constant symptom in spinal tumors. The course of the disease, steadily progressive in tumor, may aid the diagnosis when symptoms are ambiguous. Pachymeningitis cervicalis may give rise to somewhat similar symptoms in the arms; but the severe pain in the neck, the rigidity and the fixed posture, the absence of dissociated anæsthesia, and the lack of trophic disturbances will prevent any mistake in diagnosis.

Syringomyelia presents some of the features of bulbar palsy when the cavity invades the medulla and pons; but the cavity is rarely confined to the medulla and pons, and hence in syringomyelia the symptoms are not exclusively bulbar; thus a point of distinction between the two diseases is afforded.

Prognosis. The prognosis as to recovery is unfavorable; but inasmuch as the disease rarely progresses beyond a certain point it cannot be considered dangerous to life.

Treatment. There is no known remedy that will arrest the pathological process. The symptoms are to be treated as they arise—the paralysis, for instance, as in anterior poliomyelitis. The trophic disturbances may often be prevented by care, and if they occur are to be treated by rest, by mechanical appliances, or by surgical measures. The sensory loss cannot be remedied by faradic applications. It is to be remembered that the disease often comes to a spontaneous standstill; hence remedies of a constitutional kind are not to be implicitly trusted, even though they appear to arrest it.

CHAPTER XVI.

LATERAL SCLEROSIS. SPASTIC PARAPLEGIA.

History. A condition of stiffness in the legs that slowly advances to a state of paralysis, with increased reflexes and rigidity, but is not attended by sensory symptoms, though recognized by Turck in 1856, was first described by Charcot in 1865. It was carefully studied in 1873 by Seguin,[1] who named it tetanoid paraplegia. In 1875 a complete analysis of the clinical symptoms was made by Erb,[2] who ascribed them to an ascending sclerosis of the lateral columns of the spinal cord and named the affection spasmodic spinal paralysis. In the following year Charcot differentiated it more clearly from other spinal affections, naming it *tabes dorsale spasmodique*. Careful observation of cases soon developed the fact that as a symptomatic condition it appeared much more commonly as a secondary affection than as a primary disease. And, as the lesion of lateral sclerosis was ascertained to be in many cases the result of transverse lesions of the cord at a high level, and its identity with secondary descending degeneration in the lateral columns was established, many observers denied the existence of a primary lateral sclerosis and affirmed that every case, if of sufficient duration, would prove to be of a secondary nature. It was shown that the lateral tracts in the spinal cord, the sclerosis in which caused the symptoms, are made up of axones whose neurone bodies lie in the motor area of the cerebral cortex. And it was proven that any disease which affected these neurones, whether in the brain cortex or in the subcortical tracts, in the brain axis, or in the spinal cord, unilateral or bilateral, is capable of causing the symptom of spastic paralysis. It was evident, therefore, that as a symptom it might appear in many different diseases and be due to many various lesions.

It has been shown that lateral sclerosis results from:

1. Any disease in the brain that affects the motor tracts, such as tumors, softening, hemorrhagic destruction, or inflammation, with its degenerative results. Thus it is the lesion present in infantile cerebral palsy (Little's disease), whether unilateral or bilateral, and also in hydrocephalus. It may also occur in multiple sclerosis. After brain diseases it is usually unilateral It may be bilateral if the brain axis is destroyed and both motor tracts are affected.

2. Any disease in the spinal cord that involves the latera columns or cuts them off from their nutrient cells in the cortex of

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[1] New York Medical Journal, 1873.

[2] Virchow's Archiv, vol. lxx., and Berliner klin. Woch., No. 26, 1875.

the brain, such as transverse myelitis, hemorrhage in the cord, syringomyelia, combined or disseminated sclerosis, or tumors of the cord.

3. Any disease of the spine which compresses the spinal cord, such as caries, tumors of the vertebra, pachymeningitis, or aneurism.

There are, however, certain cases in which the symptoms of spastic paraplegia develop that cannot be assigned to any of these causes, since no symptoms of these diseases appear. And in a few such cases death has occurred and the autopsy has proven that the lesion was a lateral sclerosis.[1] Oppenheim, Dejerine and Soltas, Strümpell, and others have published cases. Therefore it is admitted that the disease may occur as a primary one, being a degeneration of the motor neurone, whose body lies in the brain cortex and whose axone lies in the lateral pyramidal tract. This is the cortico-spinal element of the motor tract.

It has already been shown that the spinomuscular element of this tract is subject to degeneration in chronic anterior poliomyelitis (page 223). It has been shown that in amyotrophic lateral sclerosis both cortico-spinal and spinomuscular elements are degenerated together (page 223). Hence the argument, from analogy, points to the existence of primary lateral sclerosis. Furthermore, Strümpell has shown that in certain families a tendency exists to an imperfect development of the lateral columns of the cord, and he has established the existence of a family type of lateral sclerosis. Therefore the existence of the disease, both congenital and acquired, must be admitted, having been proven by pathological observation.

In 1892 Erb differentiated from primary lateral sclerosis a second clinical form due to syphilis, and named it syphilitic spastic spinal paralysis. It usually develops within five years of the initial lesion. It differs from spastic paraplegia in the facts that disturbances in the control of the bladder and rectum occur, and that there may be slight subjective and objective disturbance of sensation. It has a slow onset and a chronic course. An analysis of the cases made by Koch[2] in 1893 and added to by Erb in 1902 has shown that the lesion is a transverse myelitis of the dorsal region of the cord, more or less incomplete, with secondary degeneration downward in the lateral columns and upward in the posterior columns. Erb is inclined to consider these degenerations as primary and due to the syphilo-toxins, but admits that they may be secondary to the transverse lesion. It will be considered under chronic myelitis, as I do not regard it as a true primary lateral sclerosis.

Etiology. The etiology of spastic paraplegia is obscure. In a few cases long marches and overexertion have been known to precede the development of the disease.

Trauma has been thought to be a cause, for some cases have developed after falls or blows on the back. It has been thought to be traceable to infectious diseases in a few cases.

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[1] See ten cases cited by Erb, Lancet, Oct. 11, 1902.

[2] Deut. Zeitschr. f. Nervenheilk., 1893, vol. iii.

Syphilis causes a condition closely allied to lateral sclerosis, and is probably a cause in some cases. But, as in tabes, the affection is usually a parasyphilitic disease and does not yield to mercurials and iodide of potassium.

Persons in middle life are most liable, the majority of cases developing between the ages of twenty and forty years. Even some of the family cases do not develop until after the age of twenty years.

Pathology. The pathology of the affection consists of a degeneration in the cortico-spinal element of the motor system, beginning in its peripheral portion, which lies in the lower part of the lateral pyramidal columns of the spinal cord. The degeneration appears to be a primary one, not of an inflammatory nature. Little by little the axones atrophy from below upward and disappear, the myelin that surrounds them is absorbed, and a secondary hyperplasia of neuroglia occurs, resulting in a sclerosis accurately limited to the distribution of the long tracts of motor function in the spinal cord. In the cervical region the anterior median columns of the cord have been found sclerosed. The association tracts of the cord appear to escape, and there is no affection of the anterior horns or of the spinomuscular element of the nervous system. The appearance of the spinal cord is not unlike that already shown in Figs. 35 and 36, that demonstrate secondary lateral sclerosis.

Symptoms. The symptoms of spastic paralysis are a very gradually increasing stiffness and rigidity of the muscles of the legs attended by an increase in the reflexes and a tendency to cramps and tremor. The disease may begin on one side, but soon becomes bilateral. The patient appreciates difficulty in all motions of the legs; he cannot step freely, he cannot go up stairs with comfort on account of great stiffness of the joints and muscles. It requires a great effort to produce slight movements, and passive motion is as difficult as active voluntary motion.

The gait is characteristic of the affection. The feet are not lifted from the ground, the toes are dragged, the shoe wears out on its inner surface and toe, the legs cannot be abducted freely, and the knees have a tendency to overlap. The patient shuffles along the ground, his steps becoming short, there being trepidation due to the increase of reflex action, causing a clonus of the foot at every step. Much fatigue is felt on walking, and the muscles often ache. Little by little the stiffness increases until the entire lower extremity appears to be moved as a mass without any motion of the ankle or knee-joints, and all efforts, such as crossing the leg, kneeling down, or kicking, are very much hampered and finally become impossible.

The muscles appear to be made of hard, tense cords, and offer resistance to any passive movement. Percussion upon the muscle or upon its tendon is immediately attended by a quick response, or even by severe twitchings, or by a marked clonus Such twitchings and spasms may occur spontaneously and the patients complain of cramps and of twitchings which often interfere with sleep. Ankle clonus appears early; a clonus is often obtainable in the toes, and pressure upon the patella or upon the adductor tendons of the thighs may elicit a clonus. Sometimes a sudden tonic spasm of the legs causes a straightening out of the limb and adduction of the thighs. The Babinski reflex appears early. The muscles of the hip are not as early or seriously affected as those of the knee and ankle, consequently the patient can walk for several years after the disease has developed and can move the thighs in bed, even when unable to walk. But as the disease goes on and the patient is finally confined to the chair or bed, contractures of the affected muscles occur, the knees are drawn up and overlapped, the heels are drawn tightly against the buttocks, and the greatest efforts of the examiner fail to produce an extension of the legs. In this condition, when the muscles cannot be actively or passively moved, they gradually atrophy from disuse until finally the legs are reduced to a skeleton appearance, the few muscles left being still contractured. During all this period there are no sensory symptoms excepting general muscular pains, and there is no disturbance of the bladder or rectum. There are no trophic changes unless toward the close of life long-continued pressure or lack of care results in the appearance of bed-sores. The electrical contractility of the muscles remains normal.

The disease may come to a standstill, as in cases reported by Erb which had been stationary for twenty to twenty-six years, or it may be a very slowly progressive one, and it is only in the last stages that any stiffness or rigidity of movement appears in the upper extremities. In fact, very often these escape entirely. When they are involved the extensors are first affected and become rigid before the flexors; the tendon reflexes are greatly exaggerated. Wrist and finger clonus is obtained, and a tremor often appears in the hands.

Diagnosis. The diagnosis of primary lateral sclerosis should only be made after a careful search for a cause (see page 277), and every effort should be made to discover some other disease to which the symptoms of spastic paraplegia are secondary. In the absence, however, of any such affection the diagnosis may be made. Hysteria sometimes causes a condition of spastic paralysis, but the general history of the patient and the course of the case, especially the rapid onset of symptoms in hysteria, will enable a diagnosis to be made.

Prognosis. The prognosis is invariably unfavorable as to recovery, but the course of the disease is so very slow that the patients may be assured of many years of usefulness after the disease is well developed. In some cases a stationary period occurs under careful régime.

Treatment. The treatment is palliative. Overexertion is to be avoided, and yet walking should not be abandoned until impossible.

The general health should be kept in good condition, and every means known to increase the nutrition of the nervous system should be employed.

These means are fully discussed in the treatment of locomotor ataxia, and what is there recommended to arrest the progress of the disease is equally applicable to the treatment of lateral sclerosis.

The symptom that gives greatest annoyance in spastic paraplegia is the spasmodic contraction of the muscles, causing jumping of the legs or sudden extensor spasm. This can often be controlled by hot baths or by the application of hot bags to the spine. It may be much relieved by the use of bromides in full doses or by bromide and chloral combined, or by the use of the coal-tar preparations, of which antipyrine is the best. Massage of the affected muscles may also give some relief if the spasm occurs at night and disturbs sleep.

CHAPTER XVII.

LOCOMOTOR ATAXIA. TABES DORSALIS. POSTERIOR SPINAL SCLEROSIS.

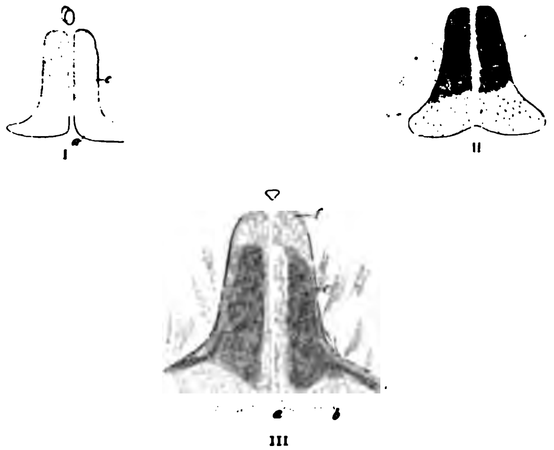
LOCOMOTOR ataxia is a chronic disease of the sensory portion of the nervous system.

Pathology. The primary lesion lies in the posterior spinal ganglia and in the ganglia of the cranial nerves. The ganglia of the sympathetic nervous system may also be involved. The neurone bodies lying in these ganglia are affected by the agent producing the disease, and consequently undergo processes of degeneration, with swelling, chromatolysis, vacuolization, pigmentation, and albuminoid and fatty degeneration of their axones.

It will be remembered that the structure of a sensory cell differs wholly from that of a motor cell (see page 23). It is a flask-shaped body with but one process. This divides at a little distance from the body into two axones, of which one passes inward through the posterior nerve root into the spinal cord or into the brain axis; the other passes outward in the nerve to the surface of the body. (See Plate I., B.) When degenerative processes attack this neurone it is possible for the degeneration to appear in the peripheral termination of the nerve at the surface of the body only. This occurs in many forms of multiple neuritis. Under these circumstances the further away the degeneration is from the body of the cell the slighter the change in the cell body, and this in many specimens escapes notice. In some cases of locomotor ataxia a degeneration of the peripheral nerves has been found. In other cases it is the central axone of the cell body, the one extending inward to the spinal cord, which appears to be primarily affected, and here again in the early stage few changes may be visible in the cell body, though the degeneration of the terminal filaments of the central axone may be complete. This is the case in locomotor ataxia in the early stage of the disease, and hence for many years the disease was supposed to be a primary posterior sclerosis, as the lesion was apparently limited to the posterior columns of the spinal cord.

But recent investigations have demonstrated that the posterior columns of the spinal cord are made up of a number of distinct systems of tracts which have been divided into two categories and named "exogenous" and "endogenous," according to the origin of the fibres taking part in their structure. The exogenous fibres are those that enter the posterior columns from without, that is, through the posterior nerve roots, and these are the fibres which are primarily involved in locomotor ataxia. Endogenous fibres are really association fibres and arise within the cord from cells of the gray matter and connect the various segments with one another. These endogenous fibres develop later in embryonal life than the exogenous fibres, and hence can be distinguished from them by the method of Flechsig. Flechsig showed that it is possible to separate various systems of fibres in the spinal cord from one another by a study of their development in fœtal life. The axones of different systems are covered by myelin at different periods. In Fig. 89 the four different systems of fibres are shown that can be distinguished from each other by his method in the posterior columns of the lumbar region of the cord. If three specimens from tabetic patients who have died in the early, middle, and last stage of the disease respectively, Fig. 90, be compared with these fœtal cords, it becomes evident that the different systems of fibres in the cord are involved in tabes at different stages of the disease, and that one system of fibres, viz., the endogenous system, always escapes.

FIG. 89.

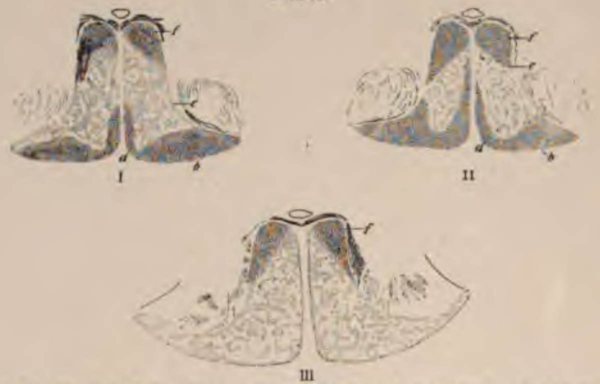


The development of the posterior columns of the spinal cord in the embryo. I. Fœtus, 24 cm. *a.* Undeveloped area. *c.* First system of fibres. II. Fœtus, 28 cm. *a.* Beginning myelinization in second system. *c.* First system completely developed. III. Fœtus, 35 cm. *a.* Partly developed second system. *b.* Partly developed third system. *c.* Fully developed first system. *f.* Partly developed fourth system. (Trepinski.)

The study of degenerations in the spinal cord occurring subsequently to lesions of the posterior nerve roots at different levels also affords a demonstration of the existence of exogenous as distinguished from endogenous fibres, the latter being unaffected in lesions of the posterior nerve roots. Sclerosis following such external lesions is identical in its situation with that occurring in the early stage of tabes. (See Figs. 44 to 46, page 189.)

For these reasons we are forced to conclude that tabes is not primarily a disease of the spinal cord, but that it is a disease of the sensory neurones, resulting in a degeneration of the exogenous fibres passing into the spinal cord from those neurones. Such degeneration, like that in other portions of the spinal cord, is followed by a condition of sclerosis, and this sclerosis is necessarily limited to the situation of the degenerated fibres.

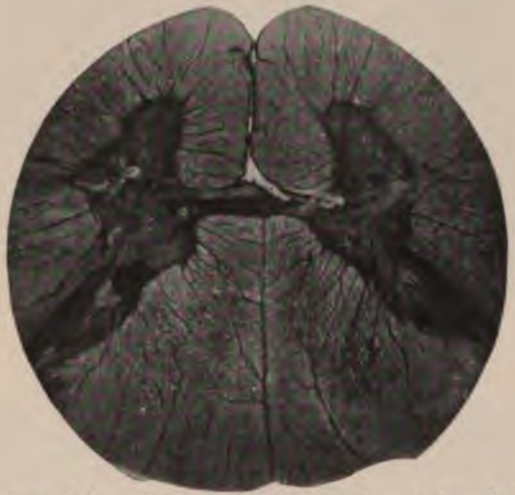
FIG. 90.



The lesions of tabes. I. The first system, *c*, is first affected by sclerosis. In the early stage, *a*, *b*, and *f* escape. II. The second, *a*, and third, *b*, systems are next involved in the sclerosis, which becomes complete in the first system, *c*, in the medium stage. III. Last stage of tabes. All systems except the fourth, *f*, are sclerotic. (Trepinski.)

It will be remembered that the posterior nerve root on entering the spinal cord consists of a number of different sets of fibres of different destination. (See Figs. 42 and 43, page 186.) Many of these fibres bifurcate on entering the cord. 1. There are fibres which enter directly at the apex of the posterior horn and turn upward in the small bundle of Lissauer and enter the posterior gray matter at a level a little higher than their entrance. Some fibres turn downward as well as upward in the column of Lissauer. 2. Other fibres enter the spinal cord opposite the posterior horn and penetrate directly into the horn, where some end in the network of fibres about the cells of the gelatinous substance or deeper within the horn, and others pass forward and cross over in the gray commissure to the opposite side, where they turn outward into the antero-lateral column or backward into the column of Goll. 3. The majority of fibres from the posterior nerve roots enter the spinal cord in what is known as the median bundle, and these pass directly into the column of Burdach, curve around the median surface of the posterior horn, and form what is known as the root zone or lateral zone of the column of Burdach. If we follow these fibres after their entrance into the root zone we find them distributed in every possible way in their passage to the posterior horn of the cord. (*a*) Some fibres turn downward, forming the comma-shaped column of Schultze, and these terminate in the second, third, and fourth segments below their point of entrance. (*b*) Others pass almost directly into the gray matter at the level of their entrance. (*c*) Others, which are short, pass upward through two or three segments. (*d*) Others still, of medium length, pass upward through four or six segments, forming the middle zone of the column of Burdach. (*e*) The remainder (long fibres) pass all the way up to the medulla oblongata, occupying the column of Goll or the median portion of the column of Burdach. An attempt is made in Figs. 42 and 43 (page 186) to demonstrate this distribution of the various fibres entering at different levels. (See also Plate XI., page 165.)

FIG. 91.



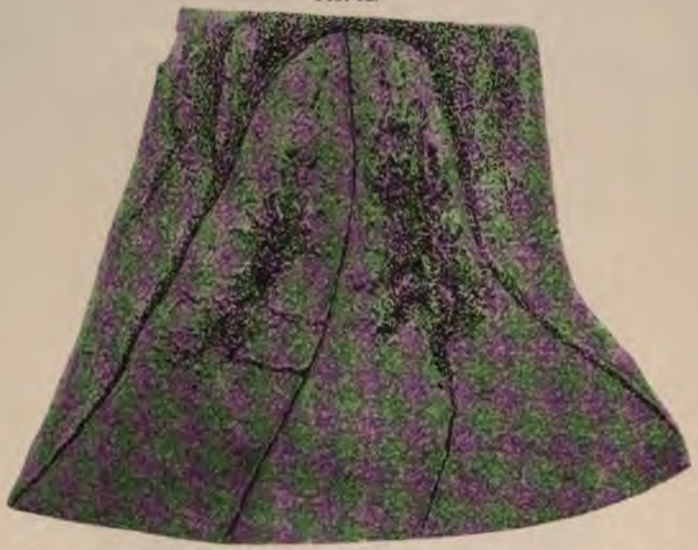
First lumbar segment of the cord in locomotor ataxia in the early stage of the disease. The sclerosis is more evident on the right side in the root zone of the column of Burdach.

All these fibres are degenerated in locomotor ataxia. The extent of the degeneration in the spinal cord will depend entirely upon the severity of the disease and upon the number of posterior nerve roots which are involved in the affection.

In the early stage of locomotor ataxia, when but few fibres are degenerated, the region of sclerosis is extremely limited in extent. As the disease begins in the vast majority of cases in the neurones of the lumbar nerves, it is in the lumbar segments only of the cord that the lesion is evident, though, inasmuch as these lumbar nerves send some fibres all the way up to the medulla, an examination will show some affection of every segment of the spinal cord at the area through which they pass. Figs. 91 and 92 demonstrate the distribution of the lesion in early cases of tabes where the lesion was thus limited to the lumbar enlargement.

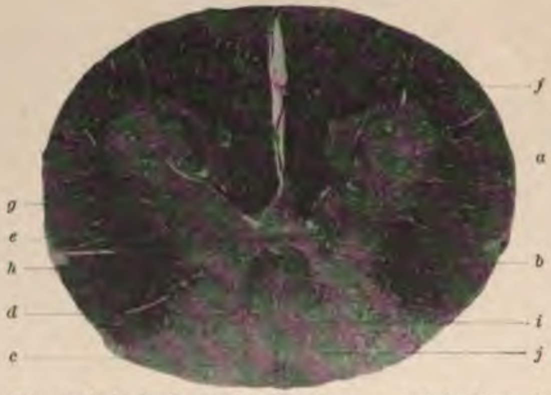
As the disease advances, a larger number of ganglia and posterior nerve roots are involved, and a greater extent of tissue is degenerated in the posterior columns. The series of sections (Figs. 93 to 95) demonstrate the lesions of tabes. The original lesion has destroyed the posterior columns in the lumbar region and has extended through the dorsal region, and involved the cervical region of the cord. Fig. 96 shows a series of sections at various levels from a patient who suffered thirty years from the disease, and whose symp-

FIG. 92.



The lesion in the root zones in the early stage of locomotor ataxia. Weigert neuroglia stain. The deeply stained tissue is sclerotic. (Schmaus-Sacki.)

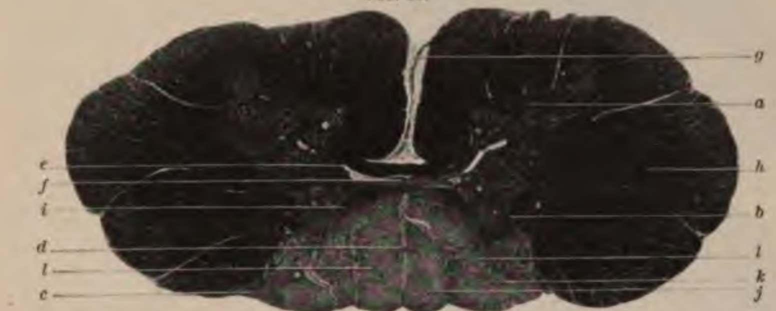
FIG. 93.



Lesion of locomotor ataxia in lumbar region, last stage. *a*. Anterior horn. *b*. Posterior horn. *c*. Posterior nerve root (sclerotic). *d*. Posterior septum. *e*. Posterior commissure. *f*. Anterior fissure. *g*. Lateral tract. *h*. Posterior commissural tract (normal). *i*. Column of Goll (sclerotic). *j*. Column of Burdach (sclerotic). (Blocq.)

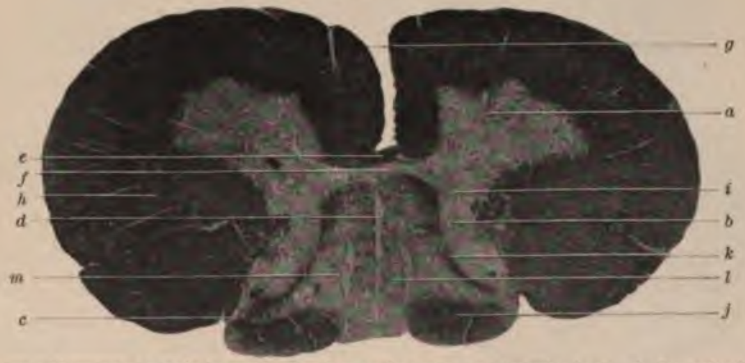
toms were as marked in the upper extremities as in the lower. In all these cases it is evident that the chief sclerosis is in the columns of Goll. The columns of Goll are made up almost entirely of fibres which have come from the sacral and lumbar regions of the spinal cord, and as these are first and chiefly affected, the lesion is most intense in them. These escape in any case of tabes limited to the cervical region.

FIG. 94.



Lesion of locomotor ataxia in cervical region, middle stage. *a.* Anterior horn. *b.* Posterior horn. *c.* Posterior nerve root. *d.* Posterior fissure. *e.* Anterior commissure. *f.* Posterior commissure. *g.* Anterior fissure. *h.* Lateral pyramidal tract. *i.* Posterior commissural tract (not affected). *j.* Column of Goll (sclerotic). *l.* Column of Burdach (partly sclerotic). (Blocq.)

FIG. 95.

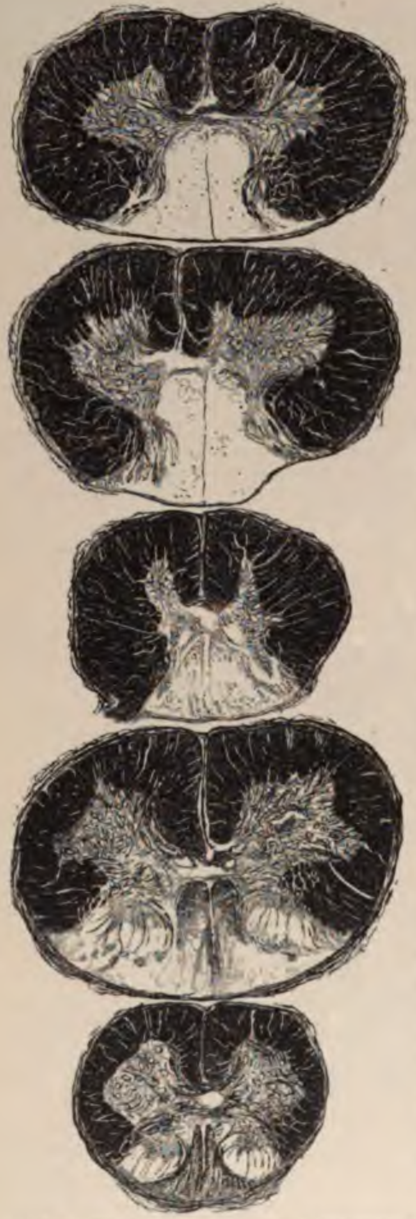


Lesion of locomotor ataxia in cervical region, early stage. *a.* Anterior horn. *b.* Posterior horn. *c.* Posterior nerve root. *d.* Posterior septum. *e.* Anterior commissure. *f.* Posterior commissure. *g.* Anterior fissure. *h.* Lateral tract. *i.* Posterior commissural tract (normal). *j.* Posterior part of column of Burdach (normal). *k.* Root zone of column of Burdach (sclerotic). *l.* Column of Goll (sclerotic). *m.* Column of Burdach, median part (sclerotic). (Blocq.)

In a case of medium intensity, when the spinal cord is removed, a thinning of the posterior nerve roots is very often apparent. They are smaller in calibre than the anterior nerve roots. The posterior surface of the spinal cord is evidently somewhat flattened in the sacral and lumbar regions, and this can be seen in the sections.

The transverse sections of the cord at different levels demonstrate the existence of sclerosis limited to the posterior columns of the cord. This sclerosis may be in early cases entirely limited to a small region adjacent to the posterior horn (Fig. 91). In other cases the sclerosis is more extensive (Fig. 96), but in all cases a region adjacent to the posterior commissure will be found to contain normal fibres, and another region adjacent to the posterior fissure and along the periphery of the cord may escape (Fig. 95). These are the posterior cornu-commissural tract and the sulco-marginal tract, which consist of endogenous fibres. A careful examination of the sclerotic region will also demonstrate the existence within it of numerous normal fibres which are also undoubtedly of endogenous origin, belonging to the third set of fibres developed in embryonal life. (See Plate XIV.)

FIG. 96.



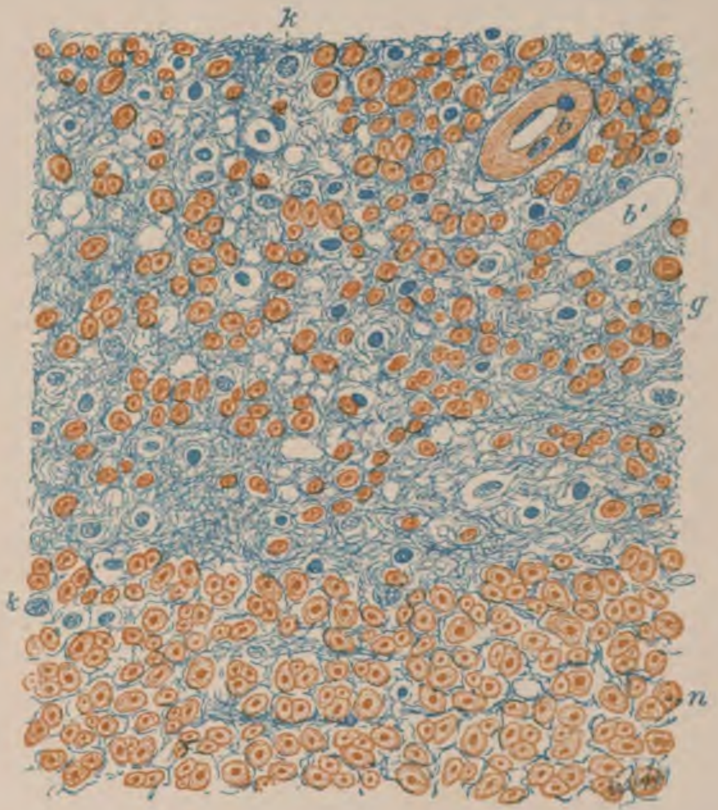
The lesions of locomotor ataxia. The sclerosis varies in degree at different levels, being more intense in the cervical region.

In very advanced cases of tabes the lesion through the cord is extensive, involving both the columns of Burdach and of Goll, but even here the escape of the cornu-commissural and sulco-marginal tracts is manifest.

Degeneration is found not only in the columns of the cord but also in the posterior horns of the cord, into which many fibres, as already stated, pass from the posterior columns. It will be remembered that the posterior horn of the cord is made up of a gelatinous and spongy substance, the substantia spongiosa, lying nearer to the periphery than the substantia gelatinosa. These two substances are permeated by the fine fibres of the cord, and these fibres are degenerated

The Gardner Sanitarium

PLATE XIV.



Sclerosis of the Posterior Column. (Schmaus.)

g. Hyperplastic glia (stained blue) with a few nerve fibres still preserved. n. Nerve fibres, yellow. k. Nuclei of the glia. b b¹. Blood-vessels. In the lower part normal fibres. Weigert's glia stain. × 850.

within the gray matter, but no special lesion can be demonstrated other than an increased pallor of these substances when methods of staining such as that of Weigert are employed.

A large number of the fibres of the posterior nerve roots pass into the column of Clarke (a column of large, round cells lying at the base of the posterior horn). These fibres are also degenerated in locomotor ataxia, and the fine plexus of terminal filaments about the cells of the column of Clarke gradually disappear. The cells of the column of Clarke themselves are not in any way altered in the disease.

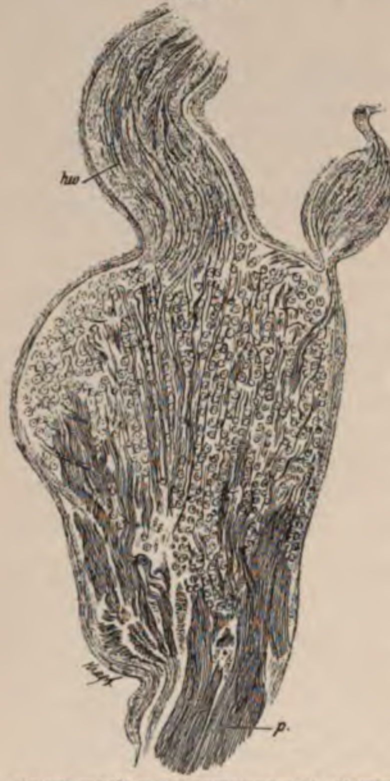
Many fibres that enter the posterior gray horn can be traced in normal cords inward through the central gray matter into the anterior horns of the cord, where they terminate about the cells of the anterior horn. These are supposed to convey impulses leading to reflex action. These fibres also degenerate in cases of locomotor ataxia; hence in advanced cases, if methods of staining are employed to demonstrate the fine network of fibres within the gray matter, this network will be seen to be thin.

The process of sclerosis present in tabes resembles that in other forms of sclerosis of the spinal cord. There is a thickening of the neuroglia tissue (Plate XIV.) and a gradual disappearance of the nerve fibres. Here and there through the glia are found spider cells and a very marked increase of glia fibres is present everywhere, as shown by the Weigert stain. Nuclear cells are distributed throughout the sclerotic patch, but this sclerosis is a process distinctly secondary to the degeneration of the nerve fibres, and may be termed a substitution hyperplasia rather than a primary formation of neuroglia.

A thickening of the meninges of the cord is commonly present in tabes, though slight in degree, and there is a closer adhesion of the meninges to the cord than in normal cords. This fact has given rise to the so-called meningeal theory of the origin of tabes, which is probably true for a certain proportion of the cases. It is supposed that a primary thickening of the meninges, either by syphilitic deposits or by a primary connective-tissue inflammation, produces a compression of the posterior roots in their passage through the meninges into the spinal cord; and hence a secondary degeneration and sclerosis occur whose distribution would naturally under these circumstances be identical with that already described. In cases where syphilis is the primary cause of a meningeal thickening a syphilitic exudation is usually present in the bloodvessels, with thickening of the intima, causing a reduction of the calibre of the vessels, and occasionally an obliteration of the lumen. This has been found in some cases of tabes. Such meningeal thickening or connective-tissue growth in the pia mater at the point of entrance of the posterior nerve roots may cause constriction at the entrance of these nerve roots and be sufficient to produce a degeneration in the nerve fibres.

Redlich[1] and Nageotte[2] have laid a great deal of stress upon this constriction as the active cause of degeneration in tabes; but it must be admitted that this is not a constant factor and that it is not present in the vast majority of the cases of tabes. Another fact which bears against this meningeal hypothesis of the origin of tabes is that in very many cases of meningitis, and even in cases of syphilitic meningitis, no such ascending degeneration as is found in tabes in the posterior root fibres can be demonstrated. Schmaus has called attention also to the fact that the absolutely symmetrical character of the degeneration in tabes would be extremely unlikely in case the origin were a diffuse meningeal process.

FIG. 97.



Spinal ganglion in locomotor ataxia, showing the degeneration in the posterior nerve root (*hw*) on the spinal side of the ganglion as contrasted with the normal appearance of the same fibres (*p*) on the peripheral side of the ganglion. (Redlich.)

Finally it is necessary to mention certain changes in the posterior spinal ganglia which have been found in tabes. An examination of the nerve roots on the peripheral and central sides of these ganglia has demonstrated that the degeneration and atrophy of the nerve fibres is found on the central side only. (See Figs. 97 to 99.) And a marked thickening of the connective tissue forming the epineurium and perineurium of these bundles of degenerated nerve fibres has been observed. This connective-tissue growth has been followed into the ganglion, and evidence of an extreme degree of interstitial inflammation has been found there. (See Fig. 100.) Whether this inflammation is the primary cause of the degeneration of the sensory neurones which lie in the ganglia is not yet determined.

While the lesion in tabes is limited to the sensory neurones of the nervous system, it must be admitted that no satisfactory theory of its pathogenesis exists. It is not yet known why the lesion is manifest in the central axone of the sensory neurone, and yet is in some cases wanting in the neurone body and in the peripheral axone.

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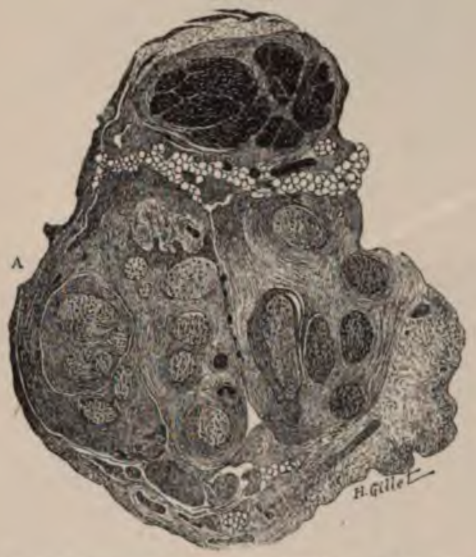
[1] Pathol. des Tabes, Jena, 1897.

[2] Bul. de la Soc. Anat., November and December, 1894

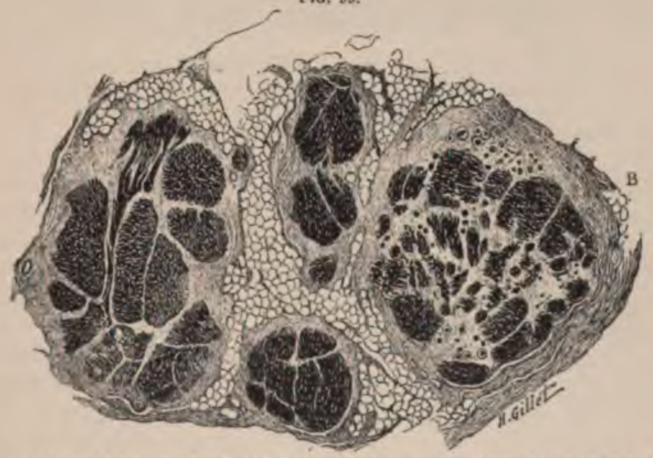
Etiology. The most common predisposing cause of locomotor ataxia is syphilis, a fact to which Erb was the first to call attention.

FIG. 98.



The lesions of the posterior nerve roots in locomotor ataxia. A. A section through the anterior and posterior nerve roots on the spinal side of the fourth lumbar posterior spinal ganglion. The anterior nerve root (above) is normal. The posterior nerve root is divided into many bundles, in which the majority of the fibres are degenerated. The increase in the connective tissue of the perineurium and epineurium in the posterior nerve root is very marked. (Thomas and Hauser, Icon. de la Salpêtrière, vol. xv., 1902, Plate XL.)

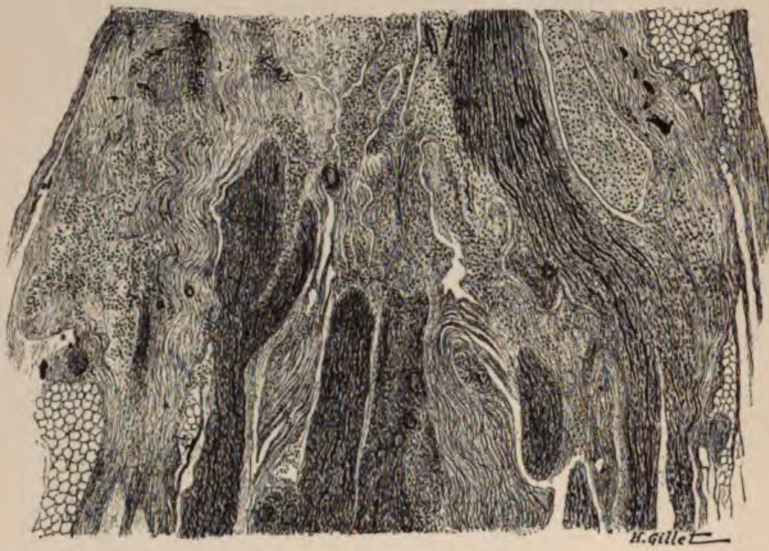
FIG. 99.



B. A section through the anterior and posterior nerve roots on the peripheral side of the same posterior spinal ganglion. Both anterior (on the left) and posterior nerve roots are normal, and the connective-tissue sheaths are not thickened. The two sections show the great difference in the posterior nerve fibres before and after their passage through the ganglion.

Various observers have published statistics which bear upon the question of the relation of syphilis to tabes. Möbius and Drummond believe that all cases are syphilitic. Erb, Fournier, Hirt, Aufimow, and Sachs found 90 per cent.; Gowers, Mendel, Senator, and Eisenlohr, 75 per cent.; Bramwell, 65 per cent.; Dana, 50 per cent.; Eulenberg and Motschutkowski, 36 per cent., syphilitic. In 127 cases in my private practice 90 were certainly syphilitic, 13 probably syphilitic, and 25 were not syphilitic; that is 86 per cent. In 332 cases in my clinic 195 were syphilitic, 39 were probably so, and 98 were not; that is 70 per cent.

FIG. 100.



Lesions of the posterior spinal ganglion in locomotor ataxia. Section through the fifth lumbar ganglion. Diminution of the fibres in the nerve roots, many of which show no myelin sheath Great increase of connective tissue throughout the ganglion, many connective-tissue nuclei, and thickening of epineurium. (Thomas and Hauser, loc. cit.)

The influence of syphilis in the production of tabes becomes more apparent when attention is called to the fact that it is present in only about 20 per cent. of cases of other forms of spinal-cord or nervous affections. Thus Erb found in 6000 cases of nervous disease, excluding tabes, that 20 per cent. were syphilitic, and Eisenkolb in 2000 cases of nervous disease that only 16 per cent. were syphilitic. We must admit, therefore, that syphilis is a very marked predisposing cause of tabes. But we cannot admit that tabes is a syphilitic disease. There are many nations and there is a class in the community (prostitutes) particularly subject to syphilis, but rather unusually exempt from tabes. Thus it is rare to find tabes in Japan, or in China, or among the negro race, though syphilis is very common in all these peoples. And almost all authorities agree that treatment by mercury and iodide of potash, while efficacious in all forms of syphilitic disease, is of little benefit in cases of tabes. Hence, while we admit that syphilis is a predisposing cause of the disease, we cannot consider tabes a syphilitic affection. The most reasonable hypothesis is that syphilis, like diphtheria, leaves in the system a toxin which has a specific action on the neurones. It may also be supposed that syphilis produces a weakened nutrition in the sensory elements of the nervous system, thus predisposing them to the deleterious action of other infections or of any agent tending to produce a malnutrition or tending in any way to exhaust the sensory elements.

There appears to be no fixed period after the syphilitic infection within which tabes develops. If a large number of cases (1000) be collected it will be found that the disease develops within five years of the infection in less than 20 per cent.; that it develops between five and ten years after the infection in 25 per cent.; between ten and fifteen years in 22 per cent.; between fifteen and twenty years in 20 per cent., and that it may develop as late as thirty or even thirty-five years after the original disease.

Exposure to cold, overexertion, especially by long-continued standing, or long marches, or a combination of these causes, such as occur in workmen who stand and labor in damp places; traumatism, such as falls or blows upon the back or upon the buttocks or upon the feet, and sexual excess are all causes of locomotor ataxia. It is to be remembered that individuals whose nutrition is poor or those who have indulged in alcoholic excesses are much more liable to develop tabes than otherwise healthy individuals. In one of my cases the symptoms followed soon after a severe blow in the dorsal region, and in a number of my cases a history of excessive standing or walking was obtained.

Locomotor ataxia is a disease of adult life. It is true that a few cases of juvenile tabes have been recorded. These are very exceptional, and have been traced in all cases to hereditary syphilis. The following table shows the age of patients affected in 1000 cases:

TABLE VII.—*Age at Onset.*

Between 10 and 20 years of age	2 cases.
" 20 " 30 " "	96 "
" 30 " 40 " "	460 "
" 40 " 50 " "	348 "
" 50 " 60 " "	90 "
" 60 " 70 " "	4 "

The disease is more common among men than among women. Of 457 cases of which I have records, 405 were males and 52 were females.

Symptoms. The symptoms of locomotor ataxia are very numerous and may be classified according to the time of their appearance. Though it must be admitted that while ordinarily there is a definite succession in the appearance of these symptoms, all cases do not conform to the rule. We distinguish between a stage of pain, a stage of ataxia, and a stage of paralysis.

The First Stage or Stage of Pain. **Paræsthesiæ** are the most common of the early symptoms of locomotor ataxia. These were complained of in all but five cases out of four hundred and fifty-two. The patient complains of tingling and numbness in the legs, of a feeling as if ants were crawling over the surface (formication), of a sensation as if the limbs were asleep, or as if he were walking upon cotton or upon pins; of unusual sensations of heat or of cold in various regions of the skin, of sensations of itching or of sharp burning, and of sensations of undue fatigue on slight exertion. These may remain during the entire course of the case and give rise to great discomfort, or as the case goes on they may subside, and in the later stages no longer be felt. They are evidence of irritation in the sensory nerve fibres, and are really illusional sensations not due to any actual disturbance in the parts in which the sensations are felt, but having their origin in the nerve roots at their entrance to the spinal cord, and being referred by consciousness not to their actual site of origin but to the periphery from which the irritated fibres arise. These abnormal sensations are usually felt first in the anterior and inner surface of the thighs or in the outer side of the legs below the knee, and gradually increase in extent until the entire lower extremities are affected. They may then be felt in the body and trunk and thorax. They finally reach the upper extremities, being felt first in the little fingers and inner half of the hand, and then in the arms or even in the neck to the head. The so-called girdle sensation is a hypersensitive condition of the skin with the production of paræsthesia on pressure. It is felt about the waist, but as the disease advances may ascend little by little until it is felt like a collar around the neck. This feeling of pressure is at times very intense and extremely disagreeable. Patients describe it as a cord or band tied tightly about the body.

Pain is the most serious and distressing of all the symptoms in locomotor ataxia. It may be the first symptom perceived; in fact, it is very often the only symptom present for many months before the physical signs of the disease appear. It was complained of in all but nine out of four hundred and fifty-two cases. This pain is of the sharp, shooting character, hence often termed "lightning pain." It is neuralgic in character, is not continuous for any length of time, but the attacks may occur with such frequency as to lead to periods of pain lasting for hours or even for days. Pain may be deep in the limbs or may be distinctly localized upon the surface of the body at some small area. It usually begins in the inner surface of the thigh, or on the anterior surface of the thigh, or about the knee, or down the outer side of the leg. As the disease advances it becomes more extensive in its distribution and advances gradually upward, being felt about the body and finally in the hands and arms. It continues as a prominent symptom throughout the first stage of the disease, but does not by any means disappear in the second stage, and though very often much less intense, it may continue into the stage of paralysis. Thus in one case at present under my care where the disease has been present for twenty years and where the patient is completely unable to walk or to stand, attacks of pain come on about twice a year, lasting for a week or ten days with as great intensity and severity as at the outset. In this patient these attacks are continuous, the sharp pain intermitting for a few minutes only and persisting night and day, preventing all rest and interfering markedly with nutrition and being attended by rapid emaciation. No special cause can be ascertained for these attacks of pain, and nothing influences them excepting enormous doses of morphine, and even ten or twelve grains a day at times fail to hold the pain in check. In this case the pains are entirely limited, as are all the symptoms, to the legs, the disease never having advanced above the level of the first lumbar segment of the cord. Sudden attacks of pain may be located about the body and in the epigastrium, attended by vomiting, and may be the first sign of the disease in certain cases. Neuralgia of the trigeminal nerve is extremely rare in tabes. Pain may be mistaken in the early stages for rheumatism or sciatica, but, as a rule, is much more limited in extent and sharper in character than rheumatic pain, and is in the early stage of the disease usually different in its distribution from the pain of sciatica. It is not to be forgotten, however, that sciatic pain may be the first sign of locomotor ataxia. The pain is different from that in multiple neuritis, inasmuch as it is not symmetrical on the two sides; it rarely affects the distal parts of the extremities first, and it is not attended by any tenderness along the nerves. Pains in the little fingers and inner side of the hand may be the first evidence that the disease has extended from the dorsal to the cervical region of the spinal cord. Pain in the back and loins of an aching character is occasionally felt, but sharp, shooting pains are not common in this locality.

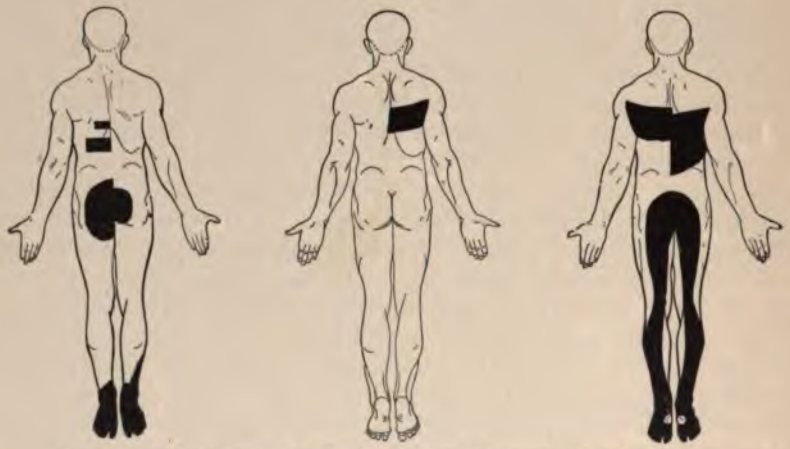
Pain is commonly attended or followed by a very hypersensitive state of the region in which it appears. This hypersensitiveness may be so great as to prevent the patient enduring the contact of clothing, or it may be more intense for temperature changes, and has been called thermal hypersensitiveness or "hypercryalgesia." It is commonly a symptom of the early stage of the disease; hence these patients are unable to endure applications of heat or cold, especially of cold, as the sensitiveness of the body to cold is markedly increased even when the sensation of heat is not affected.

Patients with locomotor ataxia are very much influenced by atmospheric conditions. This fact is to be thought of in recommending a climate in which these patients can dwell with most comfort. It is found that a low state of the barometer combined with a high percentage of humidity, either in summer or winter, is frequently followed by an attack of severe pain. It is true that the patients suffer more in cold weather than in warm, and in a cold climate more than in a warm climate; but it is the high percentage of humidity in the atmosphere, together, possibly, with certain electrical conditions not yet fully understood, which give rise to the greatest amount of pain. Thus when the humidity is over 70 per cent. and the barometer is between 29.65 and 29.90 patients are almost sure to have an attack of pain. It is also found that many patients suffer more just before a thunderstorm, just as many neurotic patients are found to have great discomfort in the form of motor restlessness and headache or disagreeable visceral and cardiac sensations prior to or during a thunderstorm. It is, therefore, to atmospheric conditions not fully understood that the sudden unexplained attacks of pain, both in tabes and in neuralgia are to be ascribed.

Pains of tabes differ from neuralgic pains rather in their distribution than in their character. The distribution of the pain in locomotor ataxia is in the region of the skin supplied by a segment of the cord. Pain in neuralgia is in the distribution of the skin supplied by definite peripheral nerves. (Compare Plates V. and XIII.)

In the stage of pain, even when paræsthesiæ, and hypersensitiveness to cold and heat are present, it is not common to discover any objective disturbance of sensation. Sometimes, however, a slight difficulty in perception of sensations of cotton-wool is admitted when the legs are compared with the hands, but it is not to be forgotten that differences of slight sensation are perceived normally in the body when the insensitive legs are compared with the more delicate hands. Laehr and Patrick have called attention to the existence of small bands of anæsthesia appearing upon the trunk, even when the legs are not anæsthetic, and Russell has demonstrated their existence on the inner surface of the arm from the axilla downward, even in the early stage of pain. Figs. 101 and 102 illustrate this condition in cases observed in my own clinic by Bonar and published by him.[1]

FIG. 101.



Areas of anæsthesia on the body and legs in cases of locomotor ataxia. (Bonar.)

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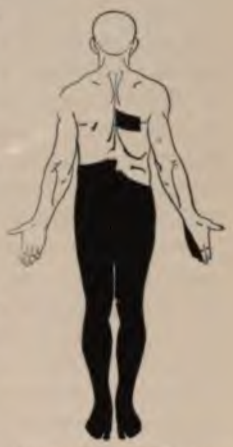
[1] Sensory Disturbances in Locomotor Ataxia. New York Medical Record, 1897, p. 721.

His examination of the clinic cases demonstrated the presence of such bands of anæsthesia in more than 90 per cent.

Disturbance in the action of the bladder and rectum is a very common symptom in the early stage of the disease. In fact, it may be the first symptom to call attention to a spinal affection. It was present in 80 per cent. of my patients. The mechanism controlling these organs lies in the sacral region of the spinal cord, but in order that this mechanism should act perfectly sensory impulses must reach the organs. Any disturbance, therefore, in the reception of sensations from the mucous membrane or muscle fibres of the bladder will produce defective action. This may be shown by an insensitiveness, so that the patient is not aware when the organ is overdistended, or it may be of the nature of abnormal sensations, causing an attempt at the emptying of the bladder when it is not at all full. A common complaint of the patients is that they cannot voluntarily empty the bladder and have to strain and press for some time before the effort is successful. Retention of urine is not uncommon in the disease, and not infrequently the bladder is never fully emptied, and hence residual urine decomposing causes cystitis. It is rare, however, for a patient with locomotor ataxia to require catheterization, and many patients find some artificial method by which they can start the act of urination. This may be by assuming an unusual position or by resorting to some unusual method. The action of the rectum is different from that of the bladder. There seems to be a loss of muscular contractile power in the involuntary muscles of the intestine, and chronic constipation is the rule in locomotor ataxia. There is a loss of expulsive power in the rectum without any relaxation of the levator or sphincter ani, hence it is usually necessary to evacuate the rectum by means of enemata.

FIG. 102.



Anæsthetic areas in a case of tabes. (Bonar.)

Impotence. An undue degree of sexual desire has been observed in the early stage of locomotor ataxia in some cases. In the majority however, there is a gradual loss of sexual power, and in the stage of ataxia the patients are usually impotent. This symptom appears about the time disturbance in the control of the bladder develops, and rarely improves under treatment. Undue attempts at coitus are often followed by an increase of the symptoms of pain and ataxia.

The **physical signs** of the disease in the early stage are the loss of patellar tendon reflex, the loss of the pupil reflex to light, and a contraction of the pupil.

The **loss of knee-jerk** is the earliest symptom of locomotor ataxia, and may give rise to a fear of the onset of this disease, even when no symptoms are present. This is particularly true of physicians. It is to be remembered that in a few normal individuals the knee-jerk cannot be elicited (2 per cent?). It is to be remembered also that in certain post-febrile conditions, noticeably after diphtheria, the knee-jerk disappears for several months. It is also to be remembered that any act of the attention directed to a spinal reflex inhibits it; hence in many individuals it is only by a diversion of the attention, by testing the knee-jerk when unexpected, or by deflection of the inhibitory impulses into other channels by means of active voluntary effort, such as clasping the hands tightly or pulling or lifting objects while the test is made, that the knee-jerk can be elicited. These methods of reinforcement of Jendrassik, as they are called, are not to be neglected in applying tests.

The knee-jerk is elicited by tapping the patellar tendon either upon its front or upon one side, when a quick contraction of the quadriceps femoris occurs, causing a slight kick. We need not enter into any discussion of the exact nature of this reflex act, as to whether it depends upon a transmission of impulses from the point irritated through the cord and outward to the muscle, or whether it is dependent upon a certain muscular tonus for whose existence sensory impressions must be acting on the centres of the spinal cord.[1] Whatever theory may be accepted, it must be admitted that a loss of tendon reflex of the knee is one of the very earliest signs of locomotor ataxia. This sign is found in 98 per cent. of the cases. It is called Westphal's sign or symptom, as it was first noticed by him. The location of the mechanism presiding over this reflex act is in the second and third lumbar segments of the cord, and these are the segments first affected by the disease; hence its value as an early sign of tabes.

The **loss of the reflex action of the pupil to light,** its contraction in the act of accommodation being preserved, was a symptom of tabes first pointed out by Argyll-Robertson, of Edinburgh. It is present in 90 per cent. of the cases. The mechanism of this reflex is still a matter of dispute, and it is not definitely ascertained whether the break in the reflex arc lies in the segment between the second and third nerves near the corpora quadrigemina or is through the sympathetic nerve, which has its origin at the first dorsal segment of the cord. It is a valuable and early sign of tabes. It may be easily elicited by covering the eyes of the patient and then suddenly exposing them to light, or by putting the patient in a dark room and with an ophthalmoscopic mirror throwing a ray of light into the eye. Not uncommonly it appears in one eye some time before it appears in the other. A continual contraction of the pupil (myosis spinalis) in which the pupil is reduced to a pin-point, does not react to light, but still reacts slightly in accommodation, is observed occasionally in the early stage of locomotor ataxia, but in the majority of cases does

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[1] Sherrington. International Medical Congress, Paris, 1900.

not develop until the later stage of the disease. In the cases in which the pains begin in the arms it is an early symptom; hence it has been referred to the lesion in the first dorsal segment of the spinal cord.

Irregular Modes of Onset. The preceding symptoms are present in the first stage of locomotor ataxia in about 85 per cent. of the cases, but in the other 15 per cent. the first stage of the disease presents certain anomalies. In fact, the disease may go on for many months without the proper diagnosis being reached, as the patient may be treated by his family physician for certain unusual symptoms or may be sent to any one of a number of specialists in eye, ear, throat, stomach, bladder, or mental disease, or even to a surgeon, for an affection of the joints or bones. It is necessary, therefore, to consider the irregular modes of onset in locomotor ataxia in the first stage of the disease. In all of these irregular modes of onset, however, a careful examination of the patient will, as a rule, reveal, either at the beginning or in the course of the distressing symptoms, the loss of knee-jerk and the Argyll-Robertson pupil. These physical signs, therefore, are essential to the diagnosis of the disease.

A very common early symptom in locomotor ataxia is the sudden development of *strabismus.* This was the first symptom in 4 per cent. of my cases. The patient notices quite unexpectedly a condition of double vision, and on looking in the glass is surprised to find that one eye is turned. The strabismus may assume any one of the various possible forms, and is occasionally attended by ptosis. It is not common for all the muscles supplied by the third nerve to be affected together, though this is possible, but the paralysis is usually limited to one or two of the muscles moving the eyeball. It is not usual for the ciliary muscle to be paralyzed. The abducens may be paralyzed, but the trochlearis always escapes. This condition of oculomotor palsy is, as a rule, very transient, lasting only a few days and passing away. In some cases it persists several weeks, but I have never known it to become permanent. Nystagmus does not occur, and I have not known oculomotor palsy to be accompanied by optic neuritis or atrophy. While these ocular palsies are often the first symptom of locomotor ataxia, it must be remembered that they may develop at any time during the course of the disease. I have seen them both in the second and third stages of the affection. It has been supposed in some cases that these palsies are of syphilitic origin and indicate exudations of gummy material upon the base or a syphilitic neuritis, such as occurs with great frequency in the oculomotor nerve and occasionally in the abducens nerve, but the fact that the oculomotor palsies of syphilitic origin subside rapidly under specific treatment, while those occurring in the course of locomotor ataxia do not so subside, would indicate that they are not of this nature.

Another symptom which may bring the locomotor ataxic patient to the oculist rather than to the neurologist is a beginning *blindness.*

This was the first symptom in 2 per cent. of my cases. It is due as a rule, to a primary optic-nerve atrophy, which condition may be the first sign of tabes, and may remain without further symptoms for many years. Thus in a case under my observation, referred to me by Knapp, the patient suffered from a gradually progressive optic atrophy which at the end of three years had rendered her totally blind, but it was not until six years after the onset of the blindness that tingling in the ulnar distribution and a loss of knee-jerks developed. There is always, therefore, in cases of primary optic atrophy a suspicion that this symptom may be the precursor of a locomotor ataxia. But in 9 per cent. of cases of locomotor ataxia of the ordinary type, with the ordinary onset, optic atrophy develops as a symptom. It may develop in the stage of ataxia or in the last stage of paralysis; hence ophthalmoscopic examinations should be made in every case where any suspicion of tabes exists. Where it is the initial symptom the patient first notices a slight diminution of clear vision and an imperfect perception of colors, and careful perimetrical examination of the visual field will demonstrate a diminution of the visual field for color and also a progressive diminution of the visual field for light. At the same time accuracy of central vision diminishes, a hazy appearance is presented to all objects, and little by little sight is lost. The ophthalmoscopic appearances are those of primary optic atrophy. (Chapter XXXIV.) Usually such an optic atrophy is bilateral. According to Erb's statistics, optic atrophy is the early symptom in 1½ per cent of the cases and develops in 18 per cent. of all cases before the end. Of 450 cases observed by me, 41 cases showed optic atrophy, and in 11 of these it was the first symptom. In cases in which it is not the first symptom of the disease, but develops subsequently, changes in the ophthalmoscopic appearance of the disk may be presented and be visible to the examiner for some time before the patient notices any diminution of sight; hence from the very outset in any case of locomotor ataxia careful examination of the visual fields as well as of the optic disks should be made. While concentric diminution of the visual field is the rule, sometimes the temporal half of the field is more diminished than the nasal half, and occasionally hemianopsia has been observed. Central scotoma is very rare.

Deafness and symptoms referable to the auditory nerve may be among the unexpected and early signs of locomotor ataxia. It is usually preceded by ringing in the ears and sometimes by attacks of vertigo, or by a constant sensation of swimming, or by difficulty in turning the head and eyes, without the development of vertigo. Examination in such cases will usually show a deafness to high notes, and a progressive diminution in the tone field will ensue, which finally results in almost total deafness. This develops usually first in one ear, but soon follows in the other. Where there is no history of hereditary deafness due to auditory atrophy, the development of such a condition should suggest the possibility of tabes. It does not develop, however, with by any means the frequency of optic atrophy. In fact, statistics show that deafness is present in but 1 per cent. of the cases.

Crises. Another mode of onset of locomotor ataxia is by the development of what are known as "crises." These were the first symptoms in eighteen of my cases and were present at some time in the course of the disease in fifty-eight cases. There are sudden attacks of imperfect function in some one of the organs of the body supplied chiefly by the pneumogastric or by the sympathetic nervous system.

The most common crisis is the gastric crisis, and patients are often treated for several years for supposed disease of the stomach and for chronic gastritis, when an examination of the eyes or of the patellar reflexes would have easily demonstrated that the cause of the symptoms was locomotor ataxia. The gastric crisis begins suddenly with severe pain in the stomach, with retching and vomiting, and this vomiting will often continue for several hours or days, all material put into the stomach being immediately rejected. Great prostration, as a rule, follows, and intense anxiety and distress. Careful examination of the gastric contents and of the gastric juice fails to reveal any constant changes. In some cases the mucous vomiting is extremely acid, in other cases there seems to be a lack of acidity. The condition, therefore, is evidently not due to any primary gastritis or disturbance of function of the glands of the stomach, but is a true tenesmus of the stomach of nervous origin. I have known it to continue for ten days, reducing the patient rapidly in weight and making it necessary to sustain life by nutritive enemata. Pain is felt constantly in the epigastrium, occasionally also between the shoulder-blades, and may encircle the body like a band. There is usually considerable tenderness in the epigastrium, the stomach rejects all food; then the act of vomiting is either futile or mucus and bile are rejected; occasionally hemorrhages occur in the stomach, and the vomited material contains so-called coffee-grounds. The vomiting is always accompanied by intense nausea, by great weakness, frequently by pains running down the arms, and oppression across the chest, by palpitation of the heart, and by vertigo. It is not uncommon for the pains of locomotor ataxia to develop in the legs during the gastric crisis, and if the crisis occurs in the course of an ordinary case these pains are usually intensified during the crisis. The agony attendant upon such a crisis is so great as to throw the patient into a state almost of delirium, in which he cries, contorts the body, and suffers all the agonies of dissolution. Hiccoughs and intestinal or rectal crises may accompany the gastric crisis.

Gastric crisis terminates suddenly, either under the influence of treatment or spontaneously, leaving the patients in a state of great prostration, but usually with increased appetite. Care has to be taken, however, in feeding them to use simple and easily digested food, in order to avoid a return of the crisis; but when all pain has ceased for twenty-four hours it is safe to begin a rapidly increasing systematic course of nourishment, as the patient needs as much food as the stomach can absorb. Forty-nine out of four hundred and fifty-six cases had gastric crises at some time during the disease. In one patient under my observation such a gastric crisis occurred at intervals, first of six months, then of three or four months, for a period of four years before a loss of knee-jerk and the development of the Argyll-Robertson pupil made it positive that the condition present was one of tabes. In the meantime he had been treated by all forms of diet, by lavage, etc., in vain. The immediate use of hypodermics of morphine in large amount on the onset of the crisis seems to cut short each attack, though they have continued to the present time at intervals, even now when the general symptoms of the disease are quite evident.

Intestinal and rectal crises are less common than gastric crises, but may also be the first signs of a locomotor ataxia. They, however, usually occur in the second stage. They begin with severe pains in the bowels or in the rectum, are attended by a watery diarrhœa with great tenesmus and rapid exhaustion, and usually by great thirst. These attacks may continue for two or three days, every attempt at taking fluid or food being followed immediately by an evacuation of the bowels. After the ordinary contents are discharged, mucus or serum is found in the discharges. These attacks cease suddenly, but leave the patient in a state of great prostration.

Laryngeal crises are next in frequency to gastric crises. The patient is usually seized by a sudden and severe cough, becomes hoarse, and has great difficulty in breathing, on account of an adductor spasm of the larynx. The cough is a typical nervous cough, very loud and harsh, is accompanied by dyspnœa, and the attacks are very severe and occur every hour or two for several minutes at a time and after a short duration are usually attended by great frequency of respiration. They may be attended by attacks of gaping. Physical examination of the lungs fails to reveal any evidence of bronchitis, but after the attack has lasted for some time inspection of the larynx usually reveals a congestion of the vocal cords, and not infrequently the intensity of the cough gives rise to a secretion of mucus. The attacks cease as suddenly as they appear, and are thus manifestly of nervous origin. But eight of my four hundred and fifty patients had laryngeal crises.

Charcot described a form of laryngeal crisis which he termed laryngeal vertigo, in which the patient feels a tickling or feeling of heat in the throat, followed by a sense of suffocation and noisy wheezing breathing. This is immediately followed by a sense of vertigo, and the patient often falls fainting and unconscious to the ground. In some persons, especially those of a very nervous constitution, Oppenheim has discovered that pressure upon the hyoid bone near to the larynx may produce reflex attacks not unlike those occurring in laryngeal vertigo and in laryngeal crisis. This fact should be remembered if patients show these symptoms, in order that they may avoid wearing a tight collar or compressing the neck.

Pharyngeal crises have been observed by Oppenheim. They consist of painful, rapidly occurring acts of swallowing, and as each act of swallowing is accompanied by the passage of a certain amount of gas into the stomach, they are followed by attacks of belching. The patient may swallow as many as twenty-four times a minute and the attack may last for ten minutes, giving rise to great distress and a sense of nausea. Attacks of hepatic colic with pain similar to that of the passage of a gallstone, and attacks of renal colic with pain similar to that of the passage of a renal calculus, occurring in the course of tabes have been described as hepatic and nephritic crises. But these are extremely rare, and their nervous origin is questionable.

Vesical and urethral crises are less common. They are attended by severe pain in the region of the bladder and in the urethra, occurring like colic in a series of sudden attacks with an intense desire to empty the bladder which may, however, not be successful. But one of my patients had this symptom.

The French authors describe genital crises in both males and females, consisting of great sexual excitement attended by sharp pains in the organs. I have never seen such cases.

Attacks of angina pectoris or pseudo-angina have given rise to the supposition that cardiac crises may occur in the course of locomotor ataxia. Patients are seized suddenly by pain in the heart and by pain running down the left arm; they turn pale, suffer much from dyspnœa and distress, with great mental anxiety, occasionally faint away, but gradually recover, though the attack may be repeated several times in twenty-four hours before it passes away completely. Such attacks are always attended by a very rapid pulse, which is not infrequently irregular and, in fact, may be preceded for several days by an unduly rapid heart action. These cardiac crises are extremely rare. I have never seen such a case.

The development of trophic disturbances in the joints and perforating ulcer of the foot rarely occurs as an early symptom of locomotor ataxia; they are much more common as complications of the later stage of the disease, and, therefore, will be considered after the symptoms of the second stage have been studied.

The Second or Ataxic Stage. The second stage of tabes has been termed the stage of ataxia and, as a rule, ataxia only develops after a preceding stage of pain or after the onset of some of the more unusual early symptoms of the disease. In a few cases (3 per cent.), however, I have seen ataxia appear as the very first symptom of locomotor ataxia. In all cases of the disease, however, it develops finally, and is one of the most important and characteristic of the symptoms of the affection, having given its name to the disease.

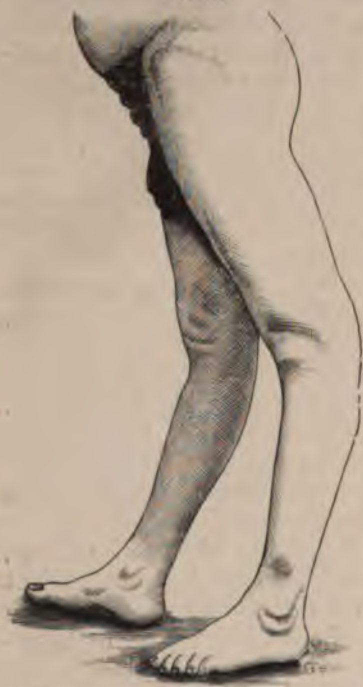
Ataxia. Ataxia may be defined as imperfect co-ordination of muscular action. For every act of the body a regular succession of movements in the muscles of proper degree and intensity and of wide distribution is necessary. The simplest act, such as closing the fist, crossing the knees, or the more complex acts of rising from a chair, standing still, taking a step, or the finer acts of writing, or buttoning the clothing, or playing a musical instrument, really involve an action in almost all the muscles of the body, for there must be a proper fixation of the joints in order that other joints may act. There must be a proper fixation of the spine in order that the balance may be preserved. There must be a proper adjustment of one side of the body in order that the other may perform its movements. Any careful study of normal action will reveal at once the highly complex process which is involved in every movement, and the length of time that is required to acquire these various adjusted movements demonstrates that many different mechanisms and combinations are required in order to produce motor effects of an adjusted character. All such movements in adult life are purely reflex and automatic, and we never think of the method pursued in the performance of a certain act unless our attention is directed to it. Even such acts as are manifestly acquired by voluntary effort, such as the learning of a new dance or the playing of a new musical instrument, soon become automatic, and while volition may set the mechanism in operation, it is the higher reflex centres that control the exact degree of muscular activity. The control of all co-ordinated movements involved in standing and walking is the function of the cerebellum. The control of all the finer movements of the hands is the function of the cerebrum; but in either case the essential condition of a proper co-ordinated act is the reception in the automatic centres, whether these lie in the spinal cord, the medulla, the cerebellum, or the cerebrum, of sensory impulses coming from the muscles, skin, and joints. In locomotor ataxia we have seen that the lesion cuts off such impulses just at their entrance into the spinal cord—the sensory tracts to the spinal cord being affected at this point—and hence impulses destined for the gray matter of the cord, for the medulla by way of the columns of Goll, for the cerebellum by way of the columns of Clarke and direct cerebellar tract, and for the cerebrum by way of the columns of Goll and Burdach, the lemniscus, and the internal capsule are all interrupted. Reference to the diagram (Plate II.) will show that a lesion of the posterior nerve roots deprives the nerve centres of all information with regard to the position of the limbs, the degree of tension of the muscles, and of all those facts which go to condition co-ordinated action and equilibrium. Hence it is to be expected that in posterior sclerosis ataxia will develop and will be exactly proportionate in degree to the degree of the sclerosis.

Ataxia is due to a loss of a large number of different sensations. It is partly due to anæsthesia of the skin, for in an ataxic patient anything which increases the anæsthesia of the soles of the feet or of the hands will intensify the ataxia. It is not wholly due to this, because ataxia does not attend anæsthesia from other causes when that is the only symptom present. Ataxia is partly due to a loss of muscular sense, the sense which probably is derived from the muscle spindles recently discovered in the muscles and tendons. This is proven by the fact that in almost all cases of ataxia a diminution, or even loss of muscular sense, can be demonstrated. This is the sense by which the situation and movements of the limbs are appreciated and by which differences of pressure and of weight are perceived. It must be clearly distinguished from the sense of effort which is a function of the cortex and is a centrifugal rather than a centripetal function. In almost all cases of ataxia careful examination will demonstrate that the patient is not sure of the exact position of his extremities or of their distal parts; cannot with his eyes closed reproduce in one hand or one foot artificial positions given to the other; cannot distinguish small differences in weights, as he should in health, and instinctively guides his movements by the sense of sight rather than by the sense of muscular feeling. But ataxia is not wholly due to the loss of this sense, for occasionally muscular sense has been obliterated, as in syringomyelia without the development of ataxia. Another important element entering into the ataxia is the sensation derived from the joints and ligaments of the joints. These sensations undoubtedly enter into the adjustment of motions, especially the sensations derived from the vertebræ, and all these sensations are undoubtedly involved in the production of true ataxia. It is therefore evident that the symptom of ataxia is due to the cutting off of numerous different forms of sensation reaching the spinal cord, but transmitted through it to the various automatic centres which control adjusted movements.

The symptom of ataxia is shown by awkwardness of motion, developing gradually and usually beginning in the legs. A patient first notices that, on closing the eyes in the act of washing, or in rising in the night and attempting to move about in the dark, he is unsteady on his feet, sways unduly, and even loses his balance. He then notices that in attempting to dance or in attempting to walk a straight line he sways unduly. Then he finds that in going up stairs, in rising from a chair, in starting off to walk, in turning suddenly while walking, or in attempting to step with precision upon a car or into a cab his feet are clumsy and he stumbles. If at this stage or in the stage of pain, even before the patient has noticed any difficulty in walking, he is asked to stand with his feet tight together and his eyes closed, he will be found to sway unduly. This is the so-called Romberg sign, having first been described by Romberg in 1852. If this act of standing with eyes closed be carefully observed it will be noticed that irregular contractions are constantly occurring in the anterior and posterior tibial muscles and in the muscles of the feet, unusual muscular effort of a wholly automatic character apparently being called into play to aid in this involuntary act.

The Gait in Locomotor Ataxia. As the disease advances the gait becomes much disturbed and a typical gait develops. The steps are irregular in their length, the feet often being placed too far apart; the legs are thrown about, their muscular act being done to excess and without proper degree. The feet are thrown forward and lifted high; they come down with a slap upon the floor, and it often seems as if the joints were too loose, the legs being thrown about with a flail-like action. In the later stages of the disease the knees are frequently bent backward in the acts both of standing and walking, and the ankles may turn. In rising from a chair such a patient is apt to lean forward unduly and sway backward and forward for a moment before gaining his proper balance. He has difficulty in starting off, usually taking hold of some adjacent object or steadying himself by a cane. These patients uniformly watch the action of their feet carefully, and many who can balance themselves fairly by the aid of eyesight are wholly unable to walk in the dark or when blindfolded. This condition is termed static ataxia in distinction from motor ataxia, which is a state of inco-ordination developing in movements not connected with standing. Any test which will involve the performance of a carefully adjusted movement will reveal the uncertainty of motion in these patients. The common test for motor ataxia is requesting the patient to follow a line on the carpet with his toe, to touch the toe to the finger of the examiner held in different positions, to touch one heel to the opposite knee, to cross the legs slowly, making the toe describe a complete circle in the air.

FIG. 103.



Abnormal position of the knees in standing in locomotor ataxia. (Dejerine.)

The hands may also become ataxic. Tests applied to the hands may be made by asking the patient to touch some object—his nose or the opposite ear—with the tip of the finger, with his eyes closed, or to perform some simple act, such as buttoning his clothing or writing. These tests will reveal the beginning ataxia in the hands when the patient perhaps is not aware of any disturbance in them. Later on, when the motor ataxia is well developed, the action of the hands is quite characteristic. The patient extends the fingers widely in making any attempt at grasping, takes hold of objects too tightly, thereby crushing delicate objects unintentionally; has great difficulty in buttoning his clothing, and the awkwardness of movement is apparent in every act of the hands. In the later stages he may be reduced to such a condition of ataxia as to be unable to write, or to feed himself, or to dress himself. If he is told to pick up a small object there may be not only an undue opening of the fingers, but irregular and unexpected jerks of the elbow and shoulder.

When the ataxia is well developed tests demonstrate an actual loss of the power of perceiving differences of weight. Thus the patient may be unable to distinguish between a penny and a half-dollar in the two hands, and when tested more accurately by means of balls of various weights will show a variation from the normal equations. The sense of pressure may also be diminished, so that when objects are piled upon the supported hand, such as two or three books, the patient will be unable to distinguish between differences that should be apparent.

Tests also will reveal a lack of knowledge of the position of the limbs in space when the eyes are closed. If a finger or toe be grasped by the examiner on two sides and movements conveyed to the joints, the patient may be unable to tell whether the finger or toe is extended or flexed. If the grasp is made on front and back of toe or finger and the pressure sense is preserved, this may convey the desired information, and the test be imperfect. This loss of the sense of position may be so extreme that the patient is unable to say which leg is placed over the other. He may lose his legs in bed and be unaware that one is hanging out and is exposed to the cold. One patient of mine always had to be carefully helped into the carriage, as he was liable to leave one leg hanging out of the door. In the most extreme form of ataxia the patient is wholly unable to stand or to walk or to use his hands, being thus apparently incapacitated for all movement; and he is then said to be in the stage of paralysis. Thus the stage of ataxia may go on slowly and merge into that of paralysis without there being any sudden transition between the two stages.

Anæsthesia. When the stage of ataxia is fully developed decided loss of sensation is usually present. The first sense to be lost is usually the sense of pain. Irritation of the skin by a pin or by a needle will develop an inability to perceive painful sensations or a very marked delay in their perception. Thus several seconds may elapse from the impact of the needle to the perception of pain, and the location of the pain may be erroneous. Usually the painful sensation is erroneously located at a level somewhat higher than that at which it really occurs. The impairment of the sense of pain leads very often to the neglect of slight injuries, especially injuries about the joints, and it is undoubtedly the cause of the development of many trophic disturbances. This lack of sense of pain may also prevent the patient from noticing the painful effects of heat or cold, even when these produce injuries to the skin. The analgesia is usually attended by thermo-anæsthesia, in which the patient loses the sensation of temperature both to heat and to cold. It is my experience that cold sensations are usually well perceived and that the patients are intensely sensitive to all cold applications, even during the well-marked stage of ataxia. But heat is very often not duly perceived, and very hot objects may seem only slightly warm; hence the patients are in danger of being burned if they are allowed to take hot baths or if any hot objects, such as bags, are laid upon the body. The sensation of temperature may also be delayed in the early stage, even when felt.

Sensations of tickling are commonly impaired in the stage of ataxia.

While anæsthesia is a rather rare symptom in the stage of pain, it is always present in some degree in the stage of ataxia. The distribution of this anæsthesia has been carefully studied of late by Laehr,[1] who has demonstrated that the anæsthetic areas correspond to the distribution of the sensory nerve roots and do not correspond to the distribution of the peripheral nerves. Thus anæsthesia is very commonly delayed on the outer surface of the legs, in the third lumbar area (Plate XIII.), in the outer half of the feet, or only on the inner half of the foot in the first sacral area. Or it may develop on the anterior surface of the thighs in the second lumbar area. It has a tendency to extend from one segmental area of the legs to another until the entire lower extremities are partially anæsthetic. The anæsthesia sometimes may appear high up on the trunk and on the inner surface of the arm from the axilla downward, even when the trunk shows no sign of anæsthesia. An insensitiveness to pressure upon the nerve trunk attends the anæsthesia, so that pressure over the peripheral nerve as it curves around the fibula, while productive of tingling in the foot, is not attended by pain; and pressure upon the ulnar nerve at the elbow, while productive of tingling in the fingers, is not attended by pain. This latter symptom was first pointed out by Biernacki.

Joint Diseases. An unusual laxity of the joints is not uncommon in the stage of ataxia. The ankles are turned unduly in walking, either in or out, the patient frequently stepping upon the side of the foot without being aware of it. The knees appear to be relaxed so far as all muscular tension about them is concerned and turned backward in the act of standing or in the act of walking. (Fig. 103.) The thighs appear to be loose, so that subluxation often appears to be imminent. The same is true of the joints of the fingers, of the wrists, and of the elbows, and I have never seen a case of well-marked ataxia in which undue extension of the joints was not easily possible

It is this relaxation of the ligaments, together with the absence of the signs of pain on the production of such unusual positions, which are probably the active causes of the development of the joint diseases of locomotor ataxia, although these joint affections are commonly termed trophic complications. They have been named Charcot joints, as he was the first to describe them. Patients very commonly do not perceive the beginning of these joint affections, and only apply to a physician when the joint is enormously swollen and full of fluid. Some traumatism is undoubtedly the active cause of the development of Charcot joints. I have seen them very commonly in clinic cases, but very rarely among the higher classes, who are not, as a rule, exposed to injuries. Among one hundred and twenty-six private patients four had an affection of the knee, two of the ankle, two of the wrist, and one of the toe. The joints affected are most frequently the knee-joints or the ankle, though the elbow and the wrist may be involved. The small joints of toes and fingers are rarely affected. Several forms of joint disease may develop, but it is to be remarked that these diseases do not follow the typical forms of arthritis and that they are not attended by pain. The first symptom is usually an effusion within the joint, which goes on rapidly until the cavity of the joint is enormously distended by fluid. Thus the knee may be twice the size of the unaffected knee, or the ankle may be twice the size of the unaffected ankle. This distention of the joint with fluid separates the articular surfaces of the bones, which thus play irregularly upon one another, and then pathological changes develop in the articular surface and in the bones themselves. The

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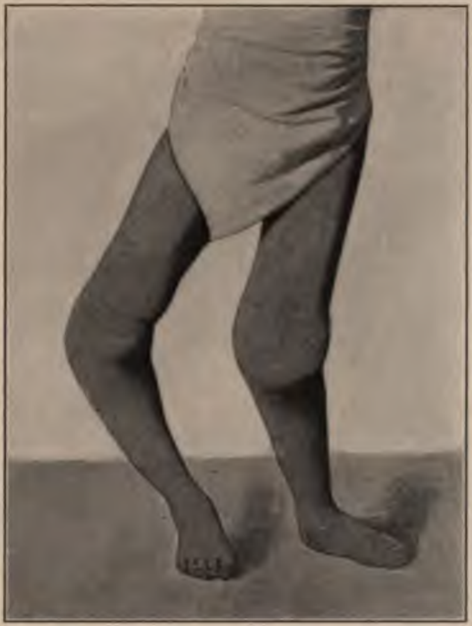
[1] Arch. f. Psych., Bd. xxvii., 688, and Bd. xxix., 648.

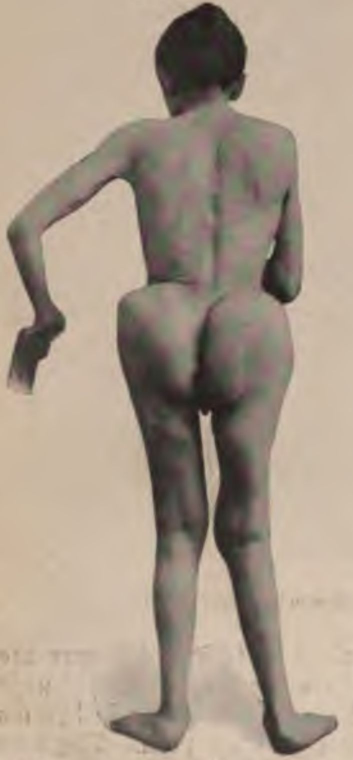
FIG. 104.



Disease of both knee-joints and both ankles in locomotor ataxia.

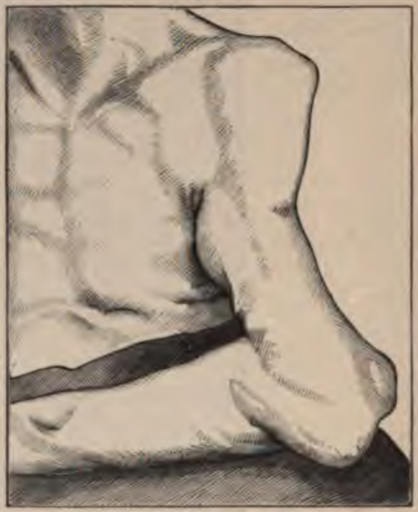
tissue all about the joint may also become infiltrated with fluid and œdematous and pit upon pressure. There is, however, no heat, no redness, no tenderness of the parts, and no pain. In mild cases effusion gradually subsides under rest and the joint returns to its normal form, cartilage and bones not having been permanently affected. In other cases an enormous thickening develops in the ends of the bones and in the cartilages, and a permanently enlarged joint that is only partly useful remains. If the process goes further this enlargement is followed by a progressive atrophy, by disintegration and disappearance of the cartilage, by erosion of the end of the bone, no trace of the articular surface finally remaining. In this last stage unusual mobility of the joints follows, and the anterior surface of the leg may be laid upon the anterior surface of the thigh without pain, or the hips may be placed in all sorts of positions without resistance. In cases where trophic disturbances of the joints occur it is not uncommon to find an undue friability of the bones, so that spontaneous fractures on very slight injury or muscular strain are produced in the long bones, especially of the lower limbs. Pathological examination has usually shown a dilatation of the Haversian canals in the long bones in these states, with a thinning and fragility of the compact substance. This has led to the general acceptance of the theory of a primary trophic disturb-

FIG. 105.



Disease of both hip-joints, with dislocation, in locomotor ataxia. (Icon. de la Salpêtrière.)

FIG. 106.



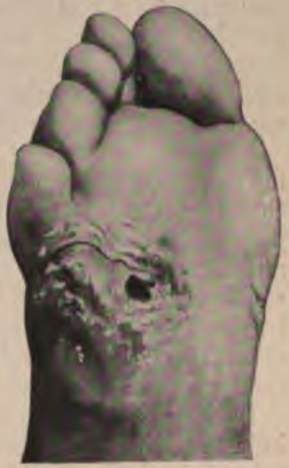
Arthropathies in shoulder and elbow in locomotor ataxia. (Dejerine.)

ance. Charcot's statement that the affection of the joints cannot be entirely explained in every case by traumatism or by neglect of painful affections must therefore be admitted.

Another trophic disturbance closely allied to joint affection is a permanent distortion of the foot which develops gradually and resembles club-foot. A hard swelling usually appears upon the back of the foot, and the inner surface of the foot becomes more prominent on account of an adducted and inverted position, the arch of the foot becomes flat, the toes are extremely flexed, the whole foot is shortened, and in attempts to walk the weight rests upon the outer surface of the foot. It is probable that this is due to a combination of factors, the lack of muscular adjustment, the relaxation of the ligaments, and a trophic disturbance of the bones of the foot coinciding to produce it.

Another trophic affection which is present in a few cases in the ataxic stage of the disease is the so-called perforating ulcer of the foot. It may originate in a neglected corn or bunion, it may be due to an injury of one of the small bones of the foot, with consequent caries and suppuration, or it may be due to a neglected hemorrhage (so-called "stone boil"). Whatever the origin, the lesion is a small sinus usually situated on the ball of the great toe, or between the great and next toe, or under the little toe, which sinus secretes a thin ichorous fluid and refuses to heal under surgical treatment. (Fig. 107.) In the vast majority of cases the absence of pain leads the patient to take no notice of the foot until the ulcer is fully formed, and then examination shows a sinus leading down to a bit of carious bone wholly insensitive and surrounded on the surface by an ulcerated dermal tissue and deeper by granulating tissue. Occasionally a gangrenous spot may form. Scraping this ulcer or its sinus, injecting it with antiseptic solutions or with iodine usually fails to be followed by healing, and occasionally, if the disease gives rise to great discomfort, which is rare, amputation of the toe or exsection of the joint may be necessary. I have known such an ulcer to be neglected by the patient, as it caused no pain for several years and produced no deleterious effects.

FIG. 107.



Perforating ulcer of the foot in locomotor ataxia. (Obersteiner.)

Peculiar skin affections are occasionally mentioned as complicating trophic disorders in the second stage of ataxia. These are usually of the form of herpetic eruptions which may be quite extensive in the legs or about the body, or of the form of subcutaneous ecchymoses, or quite widespread effusions under the skin. Pemphigus has also been observed. Lelow has described a condition of thickening of the skin with loss of color and desquamation not unlike ichthyosis. An imperfect growth of the nails with unusual ridging, peculiar curvature, or a falling out of the nails is not a very uncommon complicating trophic disturbance. Here again, however, traumatism is the usual cause.

Among trophic disturbances described loosening and falling of the teeth have been mentioned with or without severe toothache. In a few cases this condition has gone on to a necrosis of the bone of the jaw with atrophy of the alveolar process.

Irregular atrophy in the various muscles of the body, especially in the muscles supplied by the external popliteal nerve in the legs and the ulnar nerve in the hands, has been observed in a number of cases. These localized atrophies are undoubtedly due to a complicating neuritis. In the last stage of the disease, however, when the patient is confined to the bed, the entire muscular system may become atrophied. And in some of these cases a complicating amyotrophic lateral sclerosis has been found on examination of the spinal cord. The atrophy is a slowly advancing one, and is attended by paralysis and by reaction of degeneration. Sudden transient palsies have been described as occurring in tabes at any of the stages of the disease similar to the muscular palsies in the eyes. These may be due to a complicating neuritis which subsequently recovers.

Even in the stage of ataxia the muscular strength of the patient may be good, though the lack of co-ordination may make it apparent that he has a true paralysis. It is only in the last stage or stage of paralysis that any real weakness of the muscles develops. Erb affirms that in the early stage the electrical contractility of the muscles is slightly increased both to faradism and galvanism, especially in the region of the peronei, and that in the later stages there is a diminution of electrical contractility in these muscles, but never a reaction of degeneration.

It is not so easy to explain the sudden occurrence of hemiplegia in tabes, although cases have been recorded in which this has appeared both in the early and in the later stages of the disease, being temporary in character and occasionally accompanied by epileptiform convulsions. It seems likely, however, that such attacks of hemiplegia, unless developing as a symptom of general paresis, are wholly independent of the disease.

The general nutrition of the patient with tabes usually suffers in the course of the case from time to time. In the early stage a rapid loss of weight with anæmia is not at all uncommon, and, as a rule, all the symptoms of the disease are exaggerated during this period of temporary malnutrition. If, by artificial means—good food, assistants to digestion, tonics, change of climate, baths—this failing nutrition be arrested and the patient begin to gain in weight and strength, the symptoms of the disease will gradually subside, and thus it is evident that the general condition of the patient has a great deal to do with the degree of suffering that he undergoes during the course of locomotor ataxia. Hence the very great importance of supervising the care of the general health in tabetic patients, and whenever a severe onset of symptoms, either an exaggeration of pain or rapid development of ataxia, ensues, every means should be used to increase the general nutrition.

Mental Symptoms. In a certain number of cases of locomotor ataxia sudden attacks of insanity have been recorded. These usually take the form of acute maniacal excitement or of the less alarming condition of delusions of grandeur in the form of delusions of recovery from the disease, of unusual conditions of health, of unusual mental capacity, or of unusual wealth. Such insane states may last for several weeks and then gradually subside, leaving the patient in a perfectly normal state of mind with a fair recollection of what has occurred. I have watched one such case in which all the symptoms suggested paresis, but in which their disappearance in the course of three months and their failure to return during the subsequent six years convinced me that the condition was not paresis. This is a very rare complication, and must undoubtedly be ascribed to some intercurrent affection, either of a toxic character or a primary neurosis developing in the course of tabes. In a second group of cases it is the first sign of a beginning general paresis, which may go on through its typical stages and lead to dementia and paralysis. I have seen this many times. There is undoubtedly a close connection between tabes and general paresis, for not only may tabetics develop general paresis, but many paretics develop symptoms of tabes. Hence the development of mental complications should give rise to the suspicion that the tabetic patient is about to develop general paresis.

The third condition under which mental complications may arise is a condition of widespread syphilitic endarteritis of the cortex, and, inasmuch as syphilis is such a common cause of tabes, this pathological condition is to be expected. Such a syphilis of the brain may produce all the symptoms of paresis, but may yield to antisyphilitic treatment, with the result that the symptoms will gradually disappear, leading possibly to some disturbances of memory or some lack of power of self-control. It is evident, therefore, that the occurrence of mental symptoms in the course of tabes may be due to several different causes.

Rare Symptoms. A few symptoms remain to be noticed which have been recorded as developing occasionally in the course of tabes. They are certainly extremely rare.

The oculomotor palsies occasionally recur or become permanent from the outset and go on to develop all the symptoms of ophthalmoplegia externa and interna. Under these circumstances the strabismus and double vision become permanent, and ptosis is added. Very often an increase of the difficulty of walking is a result of the double vision. Some form of ocular palsy was present at some period in one quarter of my cases. It was permanent in but 5 per cent.

Hemiatrophy of the tongue has been recorded as occasionally developing in the course of tabes.

Bulbar palsy with all its distressing symptoms has been recorded as developing occasionally in tabetic patients and being the active cause of death.

Symptoms of exophthalmic goitre have been known to develop in tabetic patients, and usually have a worse prognosis than in the ordinary types of the disease, the exophthalmos being extreme, the goitre large, the tachycardia constant, and the nervous tremor of the head and body and increased sweating being very distressing to the patient.

The laryngeal crises occasionally leave a condition of permanent adductor palsy of the larynx which results in difficulty of respiration that may make tracheotomy necessary.

Muscular atrophies with paralysis may develop in almost any muscle of the body.

In a few cases a sudden rupture of the tendon Achilles has been observed, usually occurring after extraordinary efforts of the ataxic patient to preserve the balance.

Paralysis agitans has been known to develop in the course of tabes.

Many authors have pointed out the frequency of aortic insufficiency with stenosis and general arterial disease in tabetics. In one hundred and twenty-six cases I found an aortic murmur in nine. This is probably owing to the fact that syphilis is the common cause in both affections. It is hardly to be thought that there is any necessary connection between the diseases of the bloodvessels and locomotor ataxia.

Many symptoms of neurasthenia and of hysteria may develop in tabetic patients, but these are to be ascribed rather to the mental distress of long suffering than to any direct effect of the lesion in the spinal cord. Occasionally diabetes develops in the course of tabes. This again must be considered an accidental occurrence.

Stage of Paralysis. The stage of paralysis is spoken of as the third stage of tabes, but there is no hard-and-fast line between the second and third stages. When ataxia becomes so extreme as to confine the patient to his chair or couch he may be said to have entered upon the stage of paralysis. In this stage the symptoms hitherto described are exaggerated in intensity, and the danger of a fatal termination from complications becomes greater than in the second stage. As time goes on various more unusual symptoms of the disease are actively developed and the patient in the stage of paralysis presents very numerous and diverse symptoms. The chief danger of death is from a complicating cystitis or pyelonephritis; from extreme exhaustion owing to a sudden crisis of some character; from general exhaustion due to malnutrition; from bulbar paralysis, or from some intercurrent disease. As a matter of fact, very few patients die of locomotor ataxia, though cases have been recorded in which heart failure has developed and carried the patient away after a long period of tachycardia. The vast majority of patients, however, die of some intercurrent disease. And for this reason many patients do not reach the stage of paralysis, but die while in the stage of ataxia.

Course. The course of the disease in locomotor ataxia is by no means a constant one. While the majority of patients develop the first stage of pain and go on slowly after three or four years to the stage of ataxia, and remain in that stage with gradually increasing symptoms for fifteen or twenty years, then becoming paralyzed, this typical course is not conformed to in all cases. A typical history of this kind was obtained in but eighty-seven out of one hundred and twenty-six private cases.

Occasionally, after a short stage of pain or without any preceding stage of pain, the symptoms of ataxia develop very rapidly, the patient showing extreme inco-ordination of gait within three or four months of the onset of the disease. This was the history in four of my patients, and ataxia developed so rapidly as to be classed as of acute onset in six other patients. It developed very early in the disease in fourteen other cases. Hence the early appearance of ataxia may be taken as typical in about 20 per cent. of the cases. The same condition of acute ataxia may be due to an acute disseminated myelitis, in which case the further progress of the disease will fail to reveal the typical symptoms of tabes. Acute ataxia is a symptom in multiple neuritis, but the history of the causation and the analysis of other symptoms, together with the progressive course to recovery in these cases, will leave no doubt about the diagnosis.

There is a type of case in which blindness from optic atrophy develops early. The patient shows no other signs of the disease for several years, then loses his knee-jerk, develops slight lightning pains and some uncertainty of movement, but remains for many years capable of going about, and does not suffer from extreme ataxia until years have elapsed since the occurrence of the blindness. This history was obtained in nine cases.

In another class of patients gastric crises develop as an early symptom, but are found to be associated with Argyll-Robertson pupil and loss of knee-jerk, and then, after several years of paroxysmal attacks of vomiting and pain, the other symptoms of locomotor ataxia —pains, ocular palsies, and ataxia—gradually develop. This history was obtained in six cases.

In other cases, still, the occurrence of oculomotor palsies associated with optic atrophy, rapid pulse, laryngeal crises, deafness, and bulbar symptoms make it evident that almost all the cranial nerves are complicated without much affection of the spinal cord. It is true that in these cases the Romberg symptom may be present and a loss of knee-jerk may develop; but the ordinary pains and ataxia may not appear for many years after the cranial nerve palsies have rendered the patient a chronic invalid. This history was obtained in five cases.

A few cases have been described of so-called "cervical tabes" in which the symptoms of pain, numbness, and ataxia have gradually developed in hands and arms, associated with Argyll-Robertson pupil and very often with gastric crises, but in which the patients have remained for many years perfectly capable of walking about, without any pains in the legs and without any loss of knee-jerk. But one of my cases showed this mode of onset and course.

In other cases, still, the sudden occurrence of hemiplegia, of epileptiform convulsions, or of psychical disturbance of a temporary character have preceded the ordinary development of locomotor ataxia by one or two years. These symptoms, however, should not be considered as belonging to the disease locomotor ataxia and should not be considered as prodromata of tabes.

The further course of the case after the development of any one of these methods of onset is usually a slow one in locomotor ataxia. I am convinced that the disease may come to a spontaneous standstill at almost any stage. Thus I have under my observation a gentleman, aged sixty-five years, who for thirty-five years has had slight ataxic pains, slight ataxia in walking, which requires him to use a cane, but does not prevent his going about at night without it, slight disturbances in the function of the bladder, and all three physical signs of the disease. The case is clearly not a syphilitic one and has made no progress whatever during the past thirty-five years, during which time his life has been one of eminent usefulness. I have known other cases where the stage of pain has been known to last for twenty years without the development of any ataxia, all the physical signs of the disease being present and the patient expecting all the time to be incapacitated. I have known a condition of locomotor ataxia to advance rapidly and by the end of four years to have reached such a state as to render the patient incapable of walking without the assistance of two men, the ataxia of the legs being extreme. The progress then came to a spontaneous standstill; the patient has never had a single symptom above the level of the umbilicus, his ataxia has varied very much in intensity; at times he will be confined to his bed; at times he will be able to walk with the aid of one cane. He is subject about twice a year to sudden severe attacks of pain that are most agonizing and usually last from two to three weeks, during which time it is necessary for him to take from ten to fifteen grains of morphine daily, but these subside as suddenly as they have come, and, though leaving him weak for a time, do not distress him in the interval. This has been his history for fifteen years, and he has never during this time required the use of a catheter or had any gastric or intestinal disturbance. I have known the stage of ataxia to be arrested after a very slight inco-ordination has developed and the patient to remain but slightly ataxic, with few other signs of the disease, for twelve years. Under these circumstances it is necessary to be very guarded in giving a prognosis with regard to the course of a case of locomotor ataxia. Such patients are much more happy when occupied with their ordinary vocations and living as nearly a normal life as possible. Therefore, if there is no tendency to progression in the case, it is far better to discourage a life of invalidism with constant treatment and constant seeking for a favorable climate and for a new cure. It is preferable to give these patients a favorable prognosis, making them understand that they have lost certain nerve functions which cannot be regained, because of a destruction of nervous tissue, but that there is every hope that further destruction can be averted by a fair amount of care.

Diagnosis. The diagnosis of locomotor ataxia does not often present any difficulties if the history of the case is carefully studied with a knowledge of all the various methods of onset that may occur. The absolute diagnosis rests rather upon the presence of certain physical signs than upon the existence of subjective symptoms. These are the absence of contraction of the pupil to light, the loss of knee-jerk, and the swaying when the eyes are closed and feet are approximated. When any one of these symptoms is present, combined with lightning pains, with the undue fatigue after exertion, with paræsthesiæ, or with ataxia, there should be no doubt about the diagnosis. But there are certain diseases which may be mistaken for locomotor ataxia, and hence a differential diagnosis must be carefully considered. *Multiple neuritis* following alcoholism, or poisoning by arsenic, or developing subsequently to diphtheria or other infectious diseases, or developing without known cause may produce pains, ataxia, and a loss of knee-jerk, and paræsthesiæ in the limbs. But in the majority of these cases the onset of the symptoms and the development of the ataxia are far more rapid than in tabes, all the symptoms developing within three to six weeks, and the patient from being a person in fairly good health is early reduced to a stage of extreme ataxia. The history of such cases will usually enable a diagnosis to be reached. An examination of such patients demonstrates that there is no Argyll-Robertson pupil, even when there is a loss of knee-jerk, and that the symptoms are as widespread in the upper as in the lower extremities, though in tabes the hands are commonly not involved until the patient has been ataxic in the legs for several years. In peripheral neuritis the bladder and rectum are rarely, if ever, affected, while disturbance of their mechanism is an early sign in locomotor ataxia. In peripheral neuritis the distribution of the anæsthesia, when present, is in the stocking-shaped and glove-shaped areas of the skin, whereas, as already stated, in ataxia the distribution of the anæsthesia corresponds to the posterior nerve root or segment regions of the skin, and is particularly frequent upon the trunk in the form of a band, a condition never found in multiple neuritis. These points are usually sufficient to establish a differential diagnosis between these two affections, and yet I am constantly seeing cases in which there is some difficulty in distinguishing clearly between these diseases, which a careful study of the history always removes. Inasmuch as tabes is an incurable affection, while peripheral neuritis almost invariably recovers, the importance of the diagnosis, as far as prognosis is concerned, cannot be too closely insisted upon.

The diagnosis of locomotor ataxia from *general paresis* would seem to be very easy in the majority of cases, inasmuch as one is a brain and one a spinal-cord affection. But there are a few cases of general paresis which present the typical symptoms of the first stage of locomotor ataxia—lightning pains, great fatigue in the limbs, a loss of knee-jerk, and slight ataxia—and which also present the Argyll-Robertson pupil. These symptoms develop simultaneously with or soon after the mental irritability, the loss of memory, the lack of power of concentration, and the visionary and exalted ideas which are characteristic of this disease. Not infrequently patients show some disturbance of speech, tremulousness of the face, and tingling and tremor of the hands. It is in these cases that sudden transient attacks of hemiplegia are to be expected. In such cases the symptoms of tabes do not progress beyond the slight degree of ataxia and pain; do not, as a rule, play a very important rôle. It is therefore evident that one should be on the watch for the development of mental symptoms in any case of tabes, and when they occur the diagnosis of a general nervous affection in which both posterior columns of the cord and cortical lesions of the brain are present may be made. Here, again, the prognosis is important, as paresis is a disease which reaches the fatal end within three years, while tabes may remain for twenty years as a chronic affection.

The differential diagnosis between tabes and *ataxic paraplegia* is made by the existence in that disease of the symptom of spastic paralysis, stiffness and rigidity of the limbs, and an increased knee-jerk. The development of these additional symptoms will be sufficient to make it clear that a lateral sclerosis as well as a posterior sclerosis is present. Many cases of ataxic paraplegia develop the spastic symptoms with the ataxia, but the patients do not suffer from lightning pains. Both diseases are progressive and equally unfavorable in prognosis.

The diagnosis between locomotor ataxia and *disease of the cerebellum* may occasionally cause some difficulty, inasmuch as ataxia occurs in cerebellar affections. But cerebellar ataxia is wholly a static ataxia—*i. e.*, an ataxia of walking and of balancing in the upright position—and when the patient is lying down with the trunk and head supported there is no ataxia of the hands and feet. The staggering gait of cerebellar disease is much more irregular than the gait of locomotor ataxia, and it is not uncommon in cerebellar affections to find a tendency to stagger toward one side. The stamping gait with throwing of the feet high is not present in cerebellar disease. In many cases of cerebellar disease the knee-jerks are absent, but the pupil reacts to light, there are no lightning pains or numbness, and rarely any bladder disturbances.

The differential diagnosis of tabes from *disseminated sclerosis* may be made from the absence of pain in that disease, from the usual exaggeration of knee-jerk, from the prompt action of the pupils, from the presence of intentional tremor, and from the presence of nystagmus. The very many other symptoms of tabes mentioned fail to appear in the course of multiple sclerosis.

In many cases of *neurasthenia*, especially among physicians, the supposed absence of knee-jerk is liable to give rise to a fear of the existence of locomotor ataxia, and then the easy fatigue upon effort, together with the occurrence of occasional neuralgic pains and possibly slight irritability of the bladder, due to the presence of uric acid or due to the secretion of large amounts of hysterical urine, may lead the patient to suppose that the more serious disease is imminent. I have even seen an uncertainty of gait, swaying when the eyes are closed, and a complaint of a girdle sensation in such patients. But a careful examination will always demonstrate the contraction of the pupil to light; the knee-jerks will usually be elicited by the Jendrassik method or by tapping the tendon sharply when the patient is not expecting this examination, and the very slight intensity of the symptoms and the lack of development of any of the various serious symptoms of ataxia, such as gastric crises or true anæsthesia to pain, will enable a diagnosis to be reached.

Differential diagnosis between *syphilis of the spinal cord* or subacute syphilitic meningitis and locomotor ataxia is extremely difficult in some cases, for a syphilitic meningitis will cause lightning pains, paræsthesiæ, or even ataxia, and not infrequently in this affection there is a diminution or loss of knee-jerk; but the history of the case, as a rule, will give a clue to the diagnosis, for syphilitic meningitis comes on more rapidly than locomotor ataxia. Its symptoms are not as symmetrical as those in tabes; it is more likely to be attended by pain in the back and by hyperæsthesia of the trunk. The knee-jerk is rarely lost, even though diminished, and may usually be elicited by reinforcement. There is no loss of the pupil reflex to light. The pain in the back is worse at night, and bladder and rectal symptoms are usually absent.

Treatment. There are two facts to be noticed before entering upon a consideration of the treatment of locomotor ataxia:

1. The disease from its very beginning depends upon certain changes in the neurones of the spinal ganglia which are permanent in character, and hence complete recovery in any stage is impossible. All that therapeutic measures can hope to accomplish is to arrest the progress of the changes at the point reached, so that further symptoms may not develop, and this, in the majority of cases, is impossible with our present knowledge.

2. The natural history of the disease is one of very slow progress, with periods of decided remission in some symptoms and of spontaneous disappearance of other symptoms, and also with long periods during which the patient remains in a stationary condition.

Therapeutic measures may result in producing an arrest of the disease or in causing a remission of certain symptoms, yet the history of the use of numerous remedies formerly employed but now discarded, should teach us that it is often a mistake to ascribe results which may be natural to means which have been employed. It is never to be forgotten that the desire for relief and the expectation that it is to follow the use of a remedy are potent factors in producing temporary results. When, therefore, remarkable results are claimed for any new remedy in the treatment of locomotor ataxia—as, for example, electrical applications, nerve stretching, and suspension and the use of the animal extracts—the wise physician will show a becoming skepticism, even when making use of every means which may give the patient relief, and will wait for time to prove what is of actual service.

Climate. Patients suffering from locomotor ataxia are usually very susceptible to changes in the barometer and in the humidity of the atmosphere. They are more comfortable in a warm, dry climate, and any sudden change to cold or to wet weather produces an increase in their pain and inco-ordination. They also appear to suffer more in high altitudes. Therefore, it is well for those who can afford it to seek to mitigate their sufferings by residing in the South during the winter and in the North during the summer. The climate of Southern California is perhaps the most equable during the winter months, and is certainly less cold and damp than that of Florida or of the Riviera, although these latter are preferable to a northern climate. The increased susceptibility to cold makes it necessary for the comfort of the patient to keep the temperature of his house during the winter at about 70° F. and to protect him from draughts. Yet it is rarely, if ever advisable for him to abandon such an amount of out-of-door life as is most conducive to health and vigor.

Diet. It cannot be claimed that any particular form of diet is advisable in this disease. It is not to be forgotten, however, that it is of great importance to keep the patient in a good general condition and to increase, if possible, the nutrition of the body, thereby retarding the disintegration of nerve elements. Any system of diet which, by excluding certain classes of foods, tends to derange the processes of digestion and nutrition should be avoided. I have seen patients much harmed by following rigid methods of diet, such as a strictly non-nitrogenous diet, or a diet of meat, bread, and hot water, or a vegetarian diet, and, therefore, I believe that a generous diet, including all forms of food, with a slight excess of fat in the form of cream or cod-liver oil, is to be recommended. A rather free supply of water is conducive to the elimination of waste products, and the use of coffee, tea, and beer or light wines in moderation need not be forbidden, though any indulgence in spirits is to be avoided. Tobacco may also be allowed.

Exercise. From the earliest stage of the disease the patients complain that any effort, especially that of walking far or standing for a long time, produces an unusual sense of fatigue. It seems reasonable, therefore, to limit from the outset the amount of exercise taken, and, without confining the patient to the house or to his chair, it is well to caution him against any form of exertion which is sufficient to produce discomfort. Later in the course of the disease, when any movement is attended with difficulty, it may be necessary to urge upon the patient the dangers of taking to his couch or bed, and even in the stage of helplessness it is far better for him to sit up during the greater part of the day, as there is no question that the venous congestion of the spine produced by constant lying on the back is productive of an increase of pain in this disease.

Massage. During the early stage and during the last stage massage may be of service. In the early stage deep massage to the muscles of the back promotes the flow of venous blood through the spinal vessels and their anastomotic branches, and is the best means of relieving the congestion which is supposed to exist. Given at night, it often prevents the onset of paroxysms of pain, and secures sleep, especially if the patient can be taught to lie on his belly and not on his back. In the last stage, when almost all voluntary motion is impossible, general massage, by promoting venous return, gives much comfort and aids nutrition. In the long stationary stage of ataxia, when pains are not severe and when sufficient exercise can be taken to keep up a fair circulation, massage is not needed. In some cases the tenderness of the skin prevents its use at all times.

Baths. Almost every form of hydrotherapy has had its advocates, and it must be admitted that there is no method of treatment more serviceable in chronic spinal affections than the use of baths. To obtain the best results the patient must be sent to some mineral bath in the country where the combined influences of change of air, scene, surroundings, and diet, with the régime of a water-cure establishment properly conducted, can be secured. In the summer months a cool mountain resort is to be selected. In the winter a southern mild climate is to be sought. It is much to be regretted that this country offers few such establishments as are to be found in France and Germany. It is possible, however, to find in many regions the proper facilities for bathing, and well-arranged establishments are rapidly multiplying as the need is felt. The mineral constituents of the water are of much less importance than the temperature of the bath, and when the latter is correctly regulated it makes little difference whether the spring furnishes sulphur or saline or iron water.

It appears to be quite generally admitted by those who have had experience with the use of baths in this disease that tepid and warm baths are preferable to all others. Hot baths—of a temperature exceeding 97° F.—are considered injurious. They may relieve certain symptoms temporarily; in fact, no means will act more quickly than a hot bath for the relief of pain; but the ultimate effect of a succession of hot baths is to increase the rapidity of progress of the disease and to intensify the symptoms. Cold baths at a temperature below 65° Fahr. are also injurious. It seems probable that damp cold air or exposure to wet and cold are factors in the production of locomotor ataxia, and the discomfort resulting from a cold bath is very great to the majority of patients. When baths are used it is better to have the temperature of the water vary between 75° and 95° F., as within these limits a sufficient alternation of heat and cold can be secured.

There are many methods of using water in baths. Spinal douches may be given by directing a stream of water of some force from a spout against the entire length of the spine, the temperature of the water being varied during the douche from 90° F. down to 75° F., or else being kept constantly at 85° F. This may be used for ten seconds daily. Or the patient may have the spine sponged with water while he sits on the edge of a tub or in a sitz bath. The sudden alternation from heat to cold—90° to 75° F.—in such sponging may be employed at the end of the bath. Both these methods are to be followed by brisk rubbing with warm towels. A full bath at 90° F. for twenty minutes is a method frequently employed, and when a course of iodide of potassium is being pursued this is to be used daily. The "salt rubs" and "alcohol baths" of various establishments are not objectionable, provided the temperature of the water be within the limits prescribed.

It is evident that all these procedures act upon the spinal circulation, either by affecting the calibre of the vascular system in general or by producing such peripheral irritation of the skin as to cause reflex vasomotor effects. In either case the circulation in the cord is stimulated; congestion, whether arterial or venous, is relieved, and waste products removed, with the result of increasing the nutrition. Such measures may be carried on at home, provided the patient's means do not admit of the expense of a residence in an establishment.

Baths are not to be continued for very long periods consecutively. It is better for this method of treatment to be used thoroughly twice or three times a year for a period of two or three months, and then stopped. The use of tepid packs to the extremities or to the abdomen for an hour or more for the relief of pain is highly recommended. The extremity is enveloped in flannel wrung out in warm water and covered with oiled silk. The damp heat often relieves the lancinating pains promptly.

Counter-irritation. Counter-irritation to the spine, whether by blisters, cautery, setons, ice-bags, poultices, or cups, has been generally abandoned as a means of cure. In some cases pain, if severe, may be relieved by the application of an ice-bag to the spine for a short time or by the use of dry cupping. These, however, are of but temporary service, and are probably not without a harmful influence upon the progress of the lesion.

A mild form of counter-irritation to painful parts is sometimes of service in relieving the severe pain. The part may be stroked with a faradic brush, may be heated by a mustard poultice, blistering being avoided, or may be stimulated by liniment, the surface irritation appearing to counteract the pain of central origin. In all local applications in locomotor ataxia it should be remembered that great care is to be observed, for the senses of pain and temperature are often impaired, so that the patient is unable to perceive degrees of heat or pain which are harmful. If hot foot baths are used the feet should not be blistered unwittingly, and if hot bags or poultices are applied they must be watched, as no reliance can be placed on the patient's sensations.

Electricity. Electricity is an agent which has for the past fifteen years been used quite generally in the treatment of chronic spinal diseases, including locomotor ataxia. A galvanic current has been recommended by Erb and others as the only form of electricity which influences the progress of the disease. It is applied to the back, one pole being placed upon the neck or between the shoulders, the other low down upon the lumbar region. The upper pole is sometimes placed over the sympathetic ganglia in the side of the neck, though no evidence has ever been furnished of any direct action upon the sympathetic nerves, and if this method is used it is wholly empirical. A current of medium intensity is usually preferred, that is, a current not greater than twenty milliampères in strength, the electrodes employed having an area of about one hundred square centimetres (three by five inches). The direction of the current, whether ascending or descending, appears to be a matter of indifference. The duration of the application may be from three to ten minutes daily or every other day.

Those who were at first enthusiastic regarding the effects of electricity in locomotor ataxia appear to be gradually modifying their opinions, and, although this method of treatment is still pursued by some specialists, it is now discarded by many others. It is a matter of much doubt whether a current as weak as that mentioned affects the spinal cord at all. It certainly fails to produce any subjective peripheral sensations, which is the test of its reaching and affecting a peripheral nerve, such as the sciatic. On the other hand, when much stronger currents are used there seems to be some danger of unpleasant results, such as vertigo and a sudden increase of the symptoms of the disease. So little is still known of the exact effect of galvanism on the spine, and the knowledge of its action elsewhere leads so directly to the conclusion that it cannot have any effect upon the progress of a sclerosis, that it seems incumbent upon those who persist in this treatment either to bring forward some positive results or to clear themselves in some other manner of a suspicion of great credulity. For my own part, I agree with Gowers and Möbius that as a therapeutic agent in locomotor ataxia it is useless, either as a direct agent affecting the progress of the disease or as a means of treating individual symptoms.

Suspension. A recent method of treatment proposed is that of suspending the patient by his head and arms for half a minute or longer daily. This is done by means of an apparatus used by orthopedic surgeons for suspending a person during the application of a plaster jacket. The only care necessary is so to adjust the supports as to make less traction by the head than by the arms and to pad the support under the arms, so that serious compression of the nerves and veins of the axilla shall be avoided. The patient is pulled up slowly until the feet are clear of the floor. On any expression of discomfort or any appearance of syncope the process is to be stopped at once. It is thought best to begin by a short séance—one-half minute—and gradually increase the duration to three or even four minutes daily or twice daily.

This method is not considered curative, but has been said to cause relief of many symptoms, cases being recorded in which pain diminished, bladder and rectal control was re-established, sexual power was restored, and the ataxia so much relieved that walking was made more easy. It has been said that a return of the knee-jerk occurred during the treatment.

During the first year in which the method was employed reports of great improvement in many cases were published. Russell and Taylor[1] collected two hundred and fifty-five cases recorded by Continental observers, in 77 per cent. of which a distinct improvement was noted, in 30 per cent. no improvement occurred, and in 3 per cent. the patients became worse. They could not confirm these results, however, by their own experience, for out of thirty-two cases only six improved, twenty-three did not improve, and three became worse.

The experience of Erb, Bernhardt, and Dujardin-Beaumetz seems to agree with that of Russell and Taylor. My own experience in about twenty cases is even less favorable, there being but one patient out of the twenty who at the end of two years, considered it worth while to keep this treatment up. There is certainly no objection, however, to its employment. The apparatus is not expensive; it can be put up in any house, and a patient can soon learn its details sufficiently to have it carried on at home by a servant or member of his family. But the more recent reports on all sides confirm the belief that suspension, like nerve stretching and the actual cautery, is not destined to become a permanent method of treatment in locomotor ataxia. It is possible to obtain the same effect by a firm, gradual flexion of the body upon the thighs, the patient being bent forward while lying. This, like suspension, occasionally relieves pain.

Medicinal Treatment. In the majority of cases of locomotor ataxia syphilis has been the probable cause. It is not supposed that the disease is an actual manifestation of syphilis, but that it is a sequel of it. Whenever a history of previous infection is obtained, or whenever its existence is strongly suspected, it is justifiable to make a trial of specific treatment, for experience has proved that in a considerable percentage of cases this has been of benefit. Mercury is to be used by inunction, one-half drachm of the ointment being rubbed into the skin daily until a slight physiological effect is produced. It is well to mix the officinal blue ointment with an equal part of simple cerate or lanolin, as absorption is then more complete. The inunction may be made upon the back or upon various parts of the body and extremities, a new part being chosen daily. It is better absorbed if the part selected be thoroughly washed with warm water and soap and then with alcohol or ether before the application; and after the application—which should be thorough and therefore should take half an hour—a tight flannel bandage should be applied over the part rubbed; thus what is left on the surface is gradually absorbed. It aids materially in the effect if the patient takes a warm bath (95° F.) daily for twenty minutes. The production of salivation is to be avoided. If inunctions cannot be employed the mercury may be given by the stomach, the protiodide, in dose of from $\frac{1}{6}$ grain to $\frac{1}{3}$ grain three times daily, being the best preparation. The amount may be increased gradually until a slight diarrhœa is produced, and then a dose a little less than that which caused the diarrhœa may be kept up for some time. I prefer the French pills of Garnier and Lamoureux, as of uniform strength and obtainable everywhere, and I use from 3 to 12 of these pills daily. Corrosive sublimate may be employed—from $\frac{1}{60}$ grain to $\frac{1}{30}$ grain three times daily—in the same manner. The advantage of the method by inunction is that it does not affect the digestion unfavorably. Iodide of potassium is to be given in connection with the mercury in gradually increasing doses, from 15 to 100 grains three times daily. It should be administered, very largely diluted, in an alkaline water after meals, or else in a bitter tincture before meals. It is well to reduce the large dose of the iodide after a month, and then to continue it in 25-grain doses three times daily for two or three months longer. If free elimination of the iodide is secured by the use of laxatives, of diuretics (of which water is the best), and of warm baths daily, it can be taken for a long period.

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[1] Brain, 1890, vol. xiii. p. 217.

It is well to make a thorough trial of this treatment in cases of locomotor ataxia, and if a decided improvement takes place it is well to continue the iodide and to repeat the course of mercurial inunctions at intervals of four months, each mercurial course lasting from two to four weeks, according to the toleration shown by the patient. In cases in which there is a subacute spinal meningitis with the sclerosis this treatment gives the best results. It is to be remembered that the use of mercury hastens the process of optic-nerve atrophy, and therefore in the class of cases of locomotor ataxia in which optic-nerve atrophy is the initial symptom—a class quite easily separated from the ordinary type—this line of treatment is not to be employed.

In cases that are not syphilitic in origin or in which specific treatment has failed one may use other drugs. The best line of treatment, in my opinion, is the use of arsenic in small doses (arsenious acid, $\frac{1}{40}$ grain three times daily, or the cacodylate of sodium, $\frac{1}{10}$ to $\frac{1}{2}$ grain three times daily) or the continued use of corrosive sublimate, $\frac{1}{50}$ grain three times daily, or both used alternately. Arsenic is one of the best nerve tonics, and may possibly retard the disintegration of nerve fibres in the sclerotic areas of the cord. Corrosive sublimate is believed by many to retard the production of connective-tissue inflammations.

Ergot, which was formerly extensively employed, either in the form of fluid extract, $\frac{1}{2}$ drachm, or of ergotin, $\frac{1}{3}$ to 1 grain three times daily, is not, in my experience, of any permanent benefit. If it is used it should not be given continuously, but only four days in every week (Charcot).

Nitrate of silver has also been used for many years, and still has some reputation. It is to be noted that this drug has usually been given in pill made up with a vegetable extract. As a matter of fact, when thus prepared it turns into an oxide of silver within a day or two; hence it is quite likely that patients have not had the benefit of the drug. It should be administered with kaolin in a capsule. When so given it is very liable to disturb the stomach, and if kept up for any length of time discolors the gums, mucous membranes, and skin a dark blue. On the first sign of such an effect it should be stopped. Recently the use of this drug hypodermically has been urged, but it is too soon to report success.

Strychnine is universally discarded in the treatment of ataxia, excepting as a temporary remedy for the relief of incontinence of urine, in which it occasionally succeeds.

Belladonna has also been tried, but seems to have little claim to usefulness. There has not as yet been found any drug which can be said to be curative in the disease. I have never seen any benefit from the use of animal extracts, and the so-called "goat's lymph treatment" has failed to be of the slightest use in a number of my patients who have tried it.

There are certain symptoms of locomotor ataxia which require treatment. Pain is the chief one, and for this the most valuable remedies have been discovered recently in antipyrine, acetanilid, and phenacetin. Given preferably in a triturated powder in their ordinary dose and repeated two or three times a day, they succeed in quieting pain very well. I prefer to prescribe each remedy separately, using each for one week at a time and changing about, so that the dose need not be constantly increased. Salicylate of sodium in 10-grain to 20-grain doses sometimes acts when the drugs just mentioned fail. In many cases, however, recourse must be had to opium, codeine, or morphine. It is often possible to combine these drugs with others which neutralize their disagreeable effects, as in Brown-Séquard's neuralgic pill. Eventually, the hypodermic use of morphine may be resorted to, and seems to be wholly justifiable in a disease which is very painful and chronic.

The treatment of painful areas by the faradic brush, by cold or hot applications, or by cupping the spine, has already been alluded to.

Against the crises of locomotor ataxia we have but one sure remedy —viz., morphine hypodermically—and this should be used freely to relieve these distressing symptoms. Antipyrine and phenacetin have been recently recommended in the treatment of gastric crises.

The sense of stiffness which attends the stage of ataxia is frequently relieved by the use of warm baths, as already described.

The jumping of the limbs which often annoys the patient toward evening is best controlled by bromide of sodium given with Hoffman's anodyne.

The chronic constipation usually requires the habitual use of some laxative or enemata, especially if opium is being employed for the relief of pain.

Chronic cystitis, joint diseases, and ulcers of the foot must be treated according to the usual methods described in surgical textbooks.

For the optic-nerve atrophy no treatment seems to be of avail, although hypodermic injections of strychnine and the use of iodide of potassium have their adherents.

For the relief of the ataxia a method of training has been invented by Fränkel, of Berlin, which, if faithfully carried out, is usually successful. This method depends upon the fact that the act of walking, while ordinarily automatic, may be voluntarily directed. In locomotor ataxia the automatic mechanism is disturbed, but an effort of the attention combined with a progressive training of the muscles may enable one to acquire a voluntary gait which assists or takes the place of the automatic gait. The Fränkel movements consist of attempts to follow a straight line, or a curved line, or a triangle upon the floor with the tip of the toe; then, when these motions can be accurately performed by the aid of sight, further motions, such as taking a step of a definite length, going up stairs and going through various bodily exercises, are learned. These exercises should be gone through with several times a day, the patient resting for five minutes after every five minutes of exercise. While patients can acquire these motions themselves after instruction, yet it is found that they improve much more rapidly if they are personally instructed by the physician every day. As a result of such continued instruction the gait may be materially improved. I have known patients who had been unable to walk for several years to regain the power. For details of this method the reader is referred to Fränkel and Goldschneider's books on the treatment of ataxia.

Well-fitted boots with side supports to the ankle and rubber heels may assist the patient to walk.

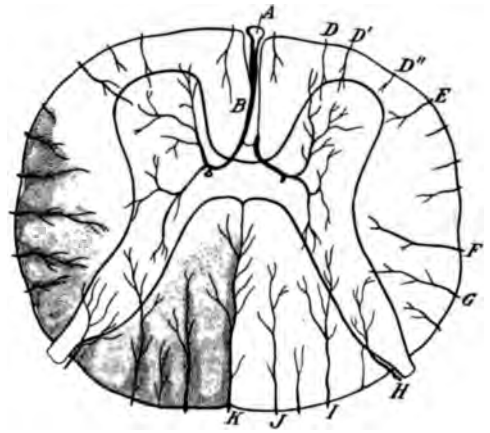
CHAPTER XVIII.

COMBINED SCLEROSIS.

Acquired and Congenital Ataxic Paraplegia. Friedreich's Ataxia.

History. Westphal, in 1867, observed a case in which both posterior and lateral columns of the cord were sclerotic. Soon after Erb, Pierret, and von Leyden recognized the fact that this may be the terminal lesion in some cases of locomotor ataxia. In 1877 Kahler and Pick and in 1878 Westphal described combined sclerosis as an independent affection. In 1886 Gowers presented a clinical picture of a disease which he named ataxic paraplegia and showed its dependence upon a sclerosis of the posterior and lateral columns of the cord. Since that time a number of similar cases have been published, especially by Strümpell, Oppenheim, Dana, and Putnam. But considerable discussion has arisen in regard to the exact nature of the disease. Some maintain that it is a "system disease" of the spinal cord, by which it is intended to imply that the lesion begins in and is limited to certain definite columns. They have seen cases in which the columns of Goll, Burdach, and the lateral pyramidal and direct cerebellar columns were affected together. Others have shown that the lesion is not strictly limited to the various functional tracts, but is much more diffuse than it is in such diseases as tabes or amyotrophic lateral sclerosis. In the cases examined by them the root zone of the column of Burdach was much less affected than the remainder of the column, the septo-marginal and cornu-commissural tracts were affected, the lateral limiting layer, the column of Gowers, and some parts of the antero-lateral column were involved; in a word, there was a diffuse sclerotic process of varying intensity at different levels of the cord, from which the anterior portions only and the anterior horns were exempt. In some of these cases there was considerable atrophy of the gray matter about the central canal and of the columns of Clarke. Hence, Leyden has recently reaffirmed that combined sclerosis is merely a form of chronic diffuse myelitis; that the distribution of the lesion through long tracts of the cord is merely due to the structure of the organ, any local lesion being necessarily followed by secondary degenerations upward or downward that are greater in extent the nearer the lesion to the periphery of the cord, since the longest fibres lie on the surface, and he denies the existence of a special disease aside from chronic myelitis. Whatever the outcome of this pathological controversy, it remains a fact that the majority of clinical observers recognize a combination of symptoms that constitute a special disease, inasmuch as they differ from other recognized diseases. And the origin of this disease appears to have been rightly disclosed by the investigations of Marie, who traces it to a vascular cause. It is well known that the spinal cord is supplied by two sets of arteries, one which enters deep within the organ through the anterior fissure and supplies its anterior half and its gray matter, the other which sends numerous branches into the lateral and posterior columns. (Fig. 108.) Any disease in these arteries causing an anæmia of the parts supplied, any thickening of the membrane upon the posterior half of the cord compressing these arteries, or any condition impairing the nutrition of the cord by blood reaching it through these vessels might produce a degeneration. And the situation of this degeneration would necessarily be in the posterior and lateral columns to a greater or less depth. This theory seems to explain both the location of the lesion and its diffuse character.

FIG. 108.



The distribution of the spinal arteries. The shaded areas are the regions which are sclerotic in combined sclerosis, and correspond to the distribution of the peripheral system of arteries. (Marie.)

Pathology. Combined sclerosis consists of a diffuse sclerotic process in the spinal cord chiefly limited to the posterior and lateral columns. In cases of moderate duration the lesion affects the column of Goll in its entire length, the column of Burdach in part (the root zone, the peripheral portion and the part adjacent to the commissure often escaping), the direct cerebellar tract, and the lateral pyramidal tract. In cases of long duration the lateral limiting layer, the tract of Gowers, and a part of the antero-lateral tract adjacent to the pyramidal tract, as well as the cells of Clarke's column and of the central gray matter, are also degenerated and the sclerosis is more complete in the columns first affected. The extent of the lesion vertically also differs in different cases, according to their duration. In the majority of cases the dorsal region of the cord is the part first involved, and as the case goes on secondary degeneration as well as the extension of the original lesion increase the vertical extent of the sclerotic process. In some cases the lateral columns are more deeply involved than in others. In some cases the posterior columns are those first and most seriously affected. But if the patient lives long enough the terminal condition is one of invasion of both columns in their entire length. The varying descriptions presented by different authors of these lesions are largely due to the omission of any reference to the duration of the disease in the different cases, Putnam being the only observer to distinguish between symptoms of long and of short standing in the same case.

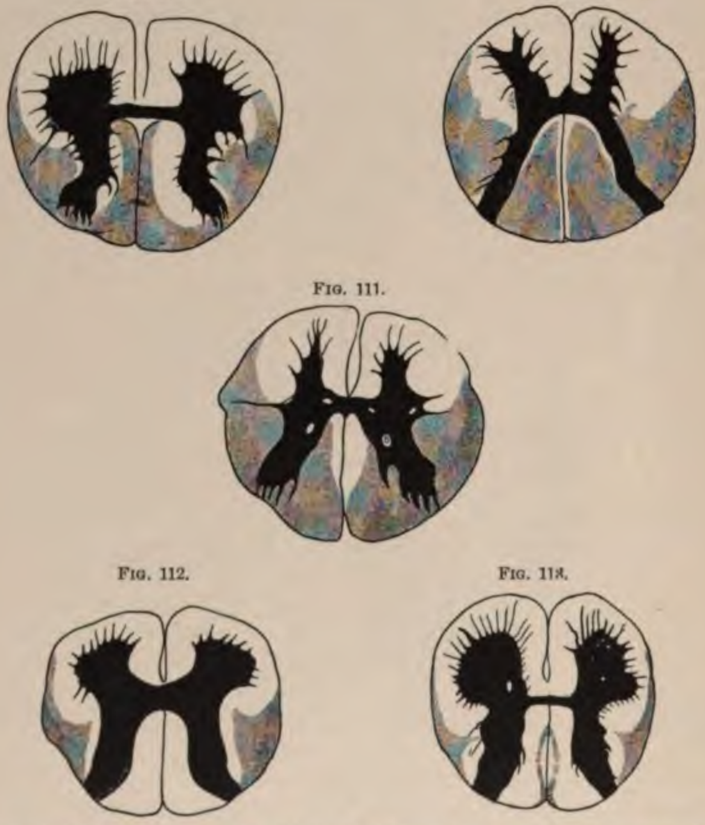


FIG. 109. FIG. 110. FIG. 111. FIG. 112. FIG. 113.

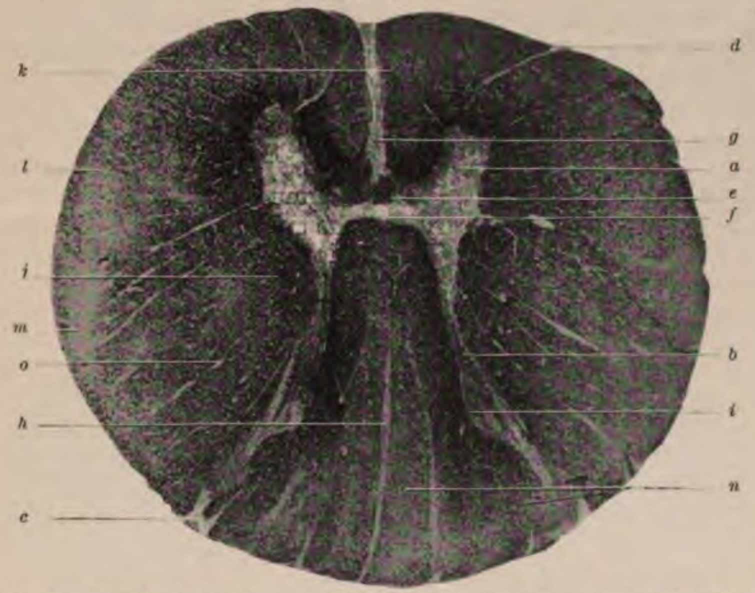
The situation of the lesion in combined sclerosis of vascular origin. Fig. 109, cervical; Fig. 110, middle dorsal; Fig. 111, lower dorsal; Fig. 112, lumbar; Fig. 113, sacral regions. In the lumbar and sacral region the lesion is one of descending degeneration only. (Ballet et Minor.)

In addition to the sclerosis of the cord the majority of observers have noticed a thickening of the pia mater, especially upon the posterior and lateral surfaces, with adhesion to the cord. Some have seen a state of arterio-capillary fibrosis or a true endarteritis in the spinal vessels. The anterior nerve roots have been found atrophied.

The combination of secondary degeneration of the motor and sensory tracts sometimes found in advanced cases of general paresis is not to be classed with this disease.

Etiology. Little is known of the cause of ataxic paraplegia, but the widespread degeneration in many columns of the cord has given rise to the supposition that it is due to an inherent want of vitality in the nervous system with a tendency to degeneration from general malnutrition of the neurones. It is certainly more common in persons of neurotic tendency. It develops usually in males in early adult life, is not particularly related to syphilis, though it has been traced to it in some cases. In many cases it follows extreme muscular exertion, and hence is more common in workmen and those subject to long marches or heavy labor. It has been known to develop after severe exhausting diseases, such as anæmia, leucocythæmia, and cancer, and especially as a complication of pernicious anæmia. Marie traces it to endarteritis in the spinal vessels.

FIG. 114.



The lesion of combined sclerosis. *a.* Anterior horn. *b.* Posterior horn. *c.* Posterior nerve root. *d.* Anterior nerve root. *e.* Anterior commissure. *f.* Posterior commissure. *g.* Anterior fissure. *h.* Posterior septum. *i.* Root zone (normal). *j.* Marginal zone (normal). *k.* Anterior column (normal). *l.* Column of Gowers (sclerotic). *m.* Direct cerebellar tract (sclerotic). *n.* Column of Goll (sclerotic). *o.* Lateral pyramidal tract (sclerotic). (Blocq.)

Symptoms. Patients suffering from combined sclerosis complain first of considerable fatigue in the legs after short walks, a sense of numbness and heaviness in the legs, and of stiffness of the muscles and of an unsteadiness of gait. This unsteadiness of gait is usually worse in the dark, and gradually as months go by the patient becomes distinctly ataxic. But this ataxia is combined with a rather marked stiffness in the action of the legs similar to that seen in lateral sclerosis; the feet are not thrown high as in locomotor ataxia, but are dragged along the ground. There is a marked swaying when the eyes are closed. There is slight diminution of tactile sensibility in the feet, less in the thighs, and there is no girdle sensation. There is an exaggeration of the knee-jerks, and ankle clonus and Babinski's reflex are present. These patients do not complain of lightning pains, as in locomotor ataxia, and they are not subject to crises. They often suffer from a dull aching sensation in the sacrum.

As the case goes on, after three or four years the spastic symptoms appear to surpass the ataxic symptoms in intensity, and spastic paraplegia, together with its characteristic gait, obscures the ataxia. These patients sometimes suffer from disturbance of the action of the bladder and rectum as the disease goes on. When the ataxia reaches the upper extremities the reflexes are increased. The Argyll-Robertson pupil is absent, but nystagmus is not infrequently developed, and this may give rise to the supposition that the disease is really disseminated sclerosis. Optic atrophy has been observed.

The course of the case is a very chronic one, but is progressive, in this respect differing from the spontaneous arrest frequently seen in locomotor ataxia. In the course of six or ten years these patients become fully paralyzed, are confined to the bed with limbs drawn up and rigid, and with all the distressing twitchings of the muscles, spasms of the legs and back, and tendency to bed-sores, which are present in cases of lateral sclerosis in the later stages. They usually die of some complication. Some cases are more rapid in their progress. This is especially true of those associated with and due to pernicious anæmia or some form of toxæmia. In these cases the final termination is reached within a year, emaciation, diarrhœa, and exhaustion preceding death. Dana and Putnam have described these cases most fully. As to the manner of onset and course of these cases there is a word to be said. The facts presented in the discussion of the pathology would indicate that the symptoms in different cases may be quite different. Oppenheim distinguishes two groups of cases: one in which the symptoms of spastic paraplegia are more prominent; the other in which the symptoms of locomotor ataxia are more marked. In the first the spastic paralysis is soon followed by the symptoms of ataxia. In the second the reverse order is seen. In my experience the latter class is more frequent than the former, and it is this class which corresponds to the ataxic paraplegia of Gowers. It is a very rare type of spinal affection.

Diagnosis. The diagnosis of the disease presents no difficulties, as is evident from the description of the symptoms. It cannot be mistaken eventually for tabes or for lateral sclerosis because symptoms of each disease appear in addition to those of the other.

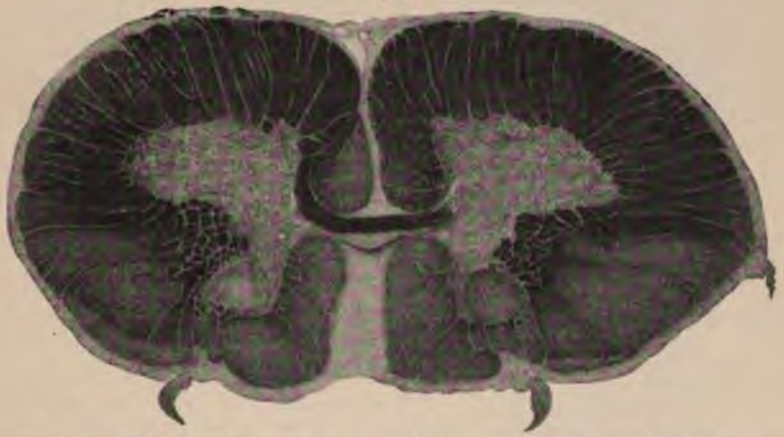
Prognosis. The disease is a slowly progressive one, and no arrest in its course can be expected. Yet occasionally periods of remission, like those occurring in locomotor ataxia, have been observed.

Treatment. Treatment is the same as that in locomotor ataxia.

FRIEDREICH'S ATAXIA.

A defective development of the spinal cord, with the production of neuroglia tissue taking the place of the defective or degenerated fibres in a number of various tracts of the spinal cord, was first described by Friedreich, of Heidelberg, in 1863 and 1876, and was named Friedreich's ataxia. Friedreich supposed it to be a juvenile form of locomotor ataxia, and called attention to its congenital origin and to the frequency with which it appeared in several members of a family. To Schultze must be given the credit of showing, in 1877, that the disease is not related in any way to locomotor ataxia, but is due to a defective development of the spinal cord. There is, apparently, a thickened and sclerotic tissue in the posterior and lateral columns of the spinal cord.

FIG. 115.



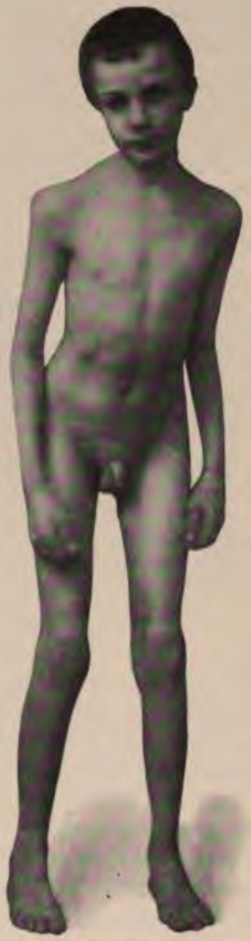
The lesion of Friedreich's hereditary ataxia. Maldevelopment and sclerosis of the lateral and posterior columns. (Schultze, Lehrbk. d. Nervenkr., Taf. iv.)

Pathology. Fig. 115, which is taken from one of Friedreich's original cases, demonstrates the peculiar appearance of the spinal cord in this condition. The spinal cord in all of these cases appears unusually thin and small, and there is usually a thickening of the pia mater about it, especially upon the posterior surface. Microscopic examination shows the presence of extensive degeneration and the remains of a few nerve fibres only in the direct cerebellar tracts, the column of Gowers, the lateral pyramidal tracts, and in the columns of Goll and Burdach. The exogenous and endogenous fibres are both involved in the lesion. The column of Goll is degenerated in its entire length and is more completely affected than the column of Burdach. The root zone of the column of Burdach and the column of Lissauer often contain very many normal fibres. The direct cerebellar column is affected as well as the pyramidal tract. In the antero-lateral column, Blocq and Marinesco have noticed a diminution in the normal number of fibres. It is evident, therefore, that a defective development in all the fibres of the spinal cord is present which is more marked in the posterior than in the anterior half. The gray matter of the cord also shows changes. There is an atrophy of the cells in both anterior and posterior horns, fewer cells being present than in a normal cord. The cells of the column of Clarke are notably degenerated and are very few in number. Occasionally around the central canal there is a thickening of the ependyma and a mass of small round cells. Both posterior and anterior nerve roots have been found somewhat atrophied. Wherever the nerve fibres are wanting, a thickening of the glia tissue is evident, but it is secondary and not primary. All authors agree that the disease is due to an arrest of development of the various systems of fibres in the spinal cord. Further investigations are necessary to confirm the statements of Marchi that secondary changes in the cerebellum are always present. Nonne and Menzel have shown, however, that an atrophy due to a defective development of the cerebellum will give rise to symptoms almost identical with those of Friedreich's ataxia. These symptoms have been best described by Marie, and it is often difficult to differentiate Friedreich's ataxia from Marie's form of cerebellar atrophy.

Etiology. The disease appears in childhood, and is sometimes present in several members of the same family; hence it has been termed hereditary. In the cases observed by me, however, but one child out of several was affected. It occasionally develops after some one of the infectious diseases of childhood, which infection is supposed to accelerate an inherent tendency to imperfect growth. In Friedreich's cases alcoholism in the parents was supposed to be the cause. Oppenheim believes that it may be a manifestation of hereditary syphilis. It may be congenital, the children never developing the power to stand or walk. It is usual, however, for the symptoms to make their appearance from the sixth to the eighth year of life, though cases are on record of as late a development as the sixteenth year. Boys are more frequently affected than girls.

Symptoms. A slow onset during childhood of symptoms which are a combination of spastic paraplegia and ataxia is characteristic of this disease. As the child learns to walk it is found that he is unsteady on his feet and awkward in his gait, being liable to fall, and he staggers in a manner suggestive of cerebellar disease. He walks slowly with little steps, unsteady, with feet far apart, and some tendency to stamp the foot, but the legs are not thrown about to the degree that is present in tabes. When the child attempts to stand he sways from side to side, the muscles of the legs contract, and the head moves as well as the body in the attempts to preserve the balance. Closing the eyes increases this unsteadiness. As the ataxia increases a certain rigidity of the legs begins to appear, and this is commonly attended by a contracture of the posterior tibial group of muscles, causing a talipes equinus. This deformity of the foot makes walking still more difficult until, little by little, the child becomes incapable of standing or walking alone. Fig. 116 shows the ordinary appearance of one of these children in standing, and Fig. 117 shows the characteristic deformity of the foot. When the child is quiet and seated constant oscillation of the body and of the head is observed, a symptom which prevented the photograph, Fig. 116, from being clear in outline. This is not a chorea-like twitching, because it is slower than the movement of chorea, and as effort to keep still increases this oscillation it reminds one of the intention tremor of disseminated sclerosis. The arms are less completely affected than the legs, but they share in the ataxia and in the intentional tremor, so that the child is soon incapacitated from feeding himself or dressing himself.

FIG. 116.



Friedreich's hereditary ataxia. The awkward posture and forward bending of the body are noticeable; also the slight flexion of knees and elbows.

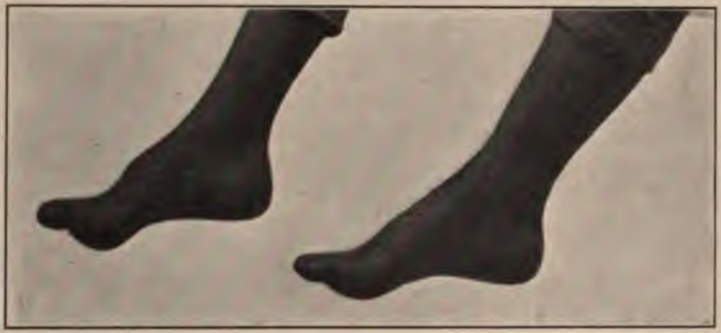
In the later stages of the affection a true paralysis may develop in the legs of the type of spastic paraplegia, the muscles being so contractured and rigid that they cannot be voluntarily moved.

Early in the disease nystagmus develops. It is not present when the eyes are quiet, but becomes visible on any attempt at turning the eyes from side to side. Paralysis of the ocular muscles, however, does not occur, and there is no tendency to optic atrophy.

A disturbance of speech is commonly present in these children. The speech is slow and difficult and indistinct, not unlike the speech of disseminated sclerosis. The tongue can be protruded, but usually shows fibrillary tremors.

There is rarely any disturbance of sensibility in the affected limbs, and shooting pains are not complained of. Sometimes the rigidity of the limbs gives rise to muscular pain. The patellar reflex is diminished or lost early in the disease, but the pupil reflex remains normal. There is no disturbance of the bladder or rectum. As a rule, the children remain rather infantile in their cerebral development. It is difficult to give them an education, and even when reaching adult life they appear to be weak-minded. But this is not always the case, though the difficulty of speech and the rather stupid appearance of the face may lead to the supposition that the child is less capable than he really is. The course of the case is a gradually progressive one. It usually appears at the age of six or seven years, rendering the child incapable and paralyzed by the age of fifteen years. It never begins after the age of sixteen years. It is not in itself a fatal affection, and therefore, if the children are well cared for they may grow up. They die of some intercurrent affection after many years of a crippled existence.

FIG. 117.



Contrast between the deformed foot in Friedreich's ataxia and a normal foot.

Diagnosis. The diagnosis of the affection is not difficult on account of the characteristic appearance of the symptoms in childhood. As children are almost exempt from locomotor ataxia, this is the only affection, excepting multiple neuritis, that will produce ataxia in children. There is no difficulty in differentiating it from multiple neuritis on account of the history of the case. Marie's cerebellar hereditary ataxia presents very similar symptoms, but this develops between the twentieth and thirtieth years; it affects the arms; the patellar tendon reflex is preserved. The characteristic contracture and deformity of the foot are not present, while, on the other hand, loss of pupil reflex, paralysis of the ocular muscles, and optic-nerve atrophy are frequently observed.

The prognosis is unfavorable, and there is no form of treatment which is known to arrest the progress of the disease.

CHAPTER XIX.

MYELITIS AND MYELOMALACIA.

Hyperæmia and Anæmia of the Spinal Cord. Lesions of Pernicious Anæmia. Acute Myelitis. Myelomalacia. Caisson Disease. Chronic Myelitis.

HYPERÆMIA AND ANÆMIA OF THE SPINAL CORD.

WHILE hyperæmia of the spinal cord may occur as an accompaniment of a general disease of a febrile character, yet as a primary affection it is questionable whether it exists, and it certainly cannot be recognized. Older writers, it is true, devoted some consideration to it, and ascribed numerous symptoms to a congestion of the spinal cord, but these symptoms are better explained at the present time as being due to neurasthenia, hysteria, or general affections of a toxic or infectious nature, and are certainly in no way referable to either arterial or venous congestion of the spinal cord.

Anæmia of the spinal cord may be said to be equally difficult of diagnosis, as it produces absolutely no characteristic symptoms. If an anæmia from obstruction in the bloodvessels is sufficiently intense to cut off the circulation in the cord, processes of softening ensue which can be recognized and which are described under the head of myelomalacia. In conditions of general anæmia, the result of long-continued malnutrition of the spinal cord is to produce degenerations of variable degree and variable extent throughout the organ. When these are sufficiently intense the symptoms caused are those of a chronic myelitis. It is very remarkable, however, that considerable degrees of degeneration have been observed after death in cases of severe anæmia when, during life, very few symptoms referable to the spinal cord have been observed. We may therefore say that both in acute and chronic anæmia of the spinal cord it is impossible to make a diagnosis of the condition; we can only suspect their presence.

PERNICIOUS ANÆMIA AND ITS EFFECTS ON THE SPINAL CORD.

There are some very marked changes produced in the nervous system by a state of pernicious anæmia,[1] and although these changes when they are slight may not produce symptoms, it is as a rule possible to detect in these cases evidence of disease of the spinal cord.

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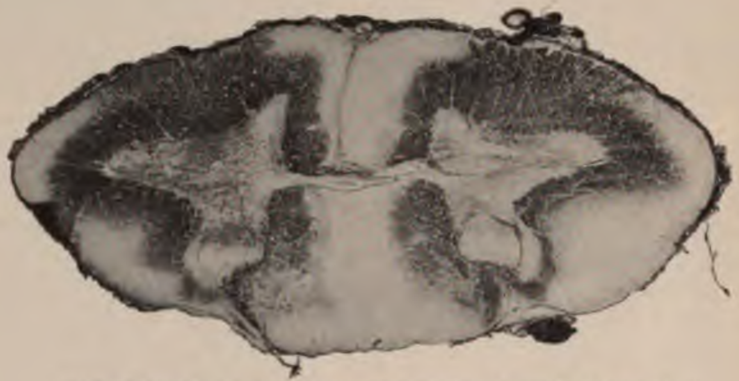
[1] Frank Billings. The Shattuck Lecture, 1902. Putnam and Taylor, Journal of Nervous and Mental Disease, 1901, vol. xxviii. pp. 1 and 74.

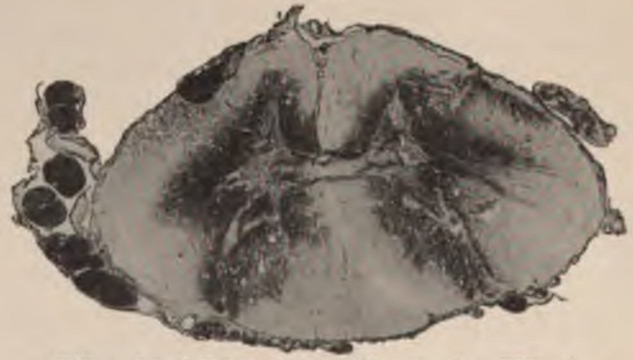
Pathology. A diffuse sclerosis has been found in all the cases reported. This is not limited to any one system of fibres, but involves any or all of the white columns. As a rule it is more intense in the posterior columns, and more marked in the cervical and upper dorsal segments of the cord than elsewhere; but the lateral and anterior columns do not escape, and the entire length of the cord may be affected. Figs. 118 to 120 demonstrate the lesion in cases reported by Billings. The sclerosis is not always diffuse, but may occur in focal areas not symmetrically distributed. It resembles multiple sclerosis in not causing secondary degenerations in the various tracts. Thus in the case shown in Fig. 119, where the lateral pyramidal tracts were sclerotic in the upper dorsal region,

FIG. 118.



Diffuse sclerosis of the cervical region in pernicious anæmia. (Billings.)

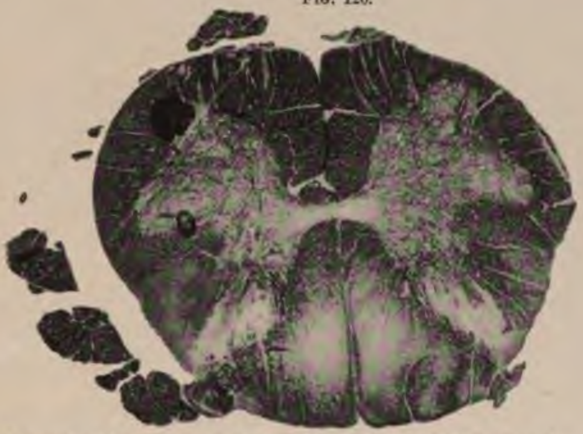
FIG. 119.



Diffuse sclerosis of the cord in pernicious anæmia. (Billings.)

they escaped in the lumbar region. (Fig. 120.) This is due to the fact that in some cases there is a degeneration in the myelin only, the axone being unaffected. The gray matter of the cord is not usually involved. Some pigmentation of the neurone bodies has been seen, but chromatolysis and degeneration have not been found. The posterior ganglia also escape degeneration.

FIG. 120.



Sclerosis of the posterior columns of the cord in pernicious anæmia. (Billings.)

Symptoms. The general symptoms of pernicious anæmia precede the nervous symptoms for several months. The patients complain first of numbness in the legs and arms, attended by a girdle sensation and a slight loss of sensation. Imperfect control of the bladder and rectum follows, sexual power is impaired, and the limbs become weak, and reflexes are lost. Then a general paralysis develops, with some emaciation, but without atrophy or electrical changes in the muscles. Sometimes an ataxic gait precedes the paralysis; sometimes the gait is spastic rather than ataxic. The course of the disease is slow, with marked remissions in the symptoms. In lighter cases, in which the disease has been arrested, all the nervous manifestations may subside. In severe cases the patients become paralyzed before the disease terminates fatally. The diagnosis is to be made from the examination of the blood.

Treatment. The treatment must be directed to the underlying condition of pernicious anæmia, by food, tonics, and the use of bone-marrow and of arsenic. This is not often successful, and the percentage of recovery from pernicious anæmia is below twenty. The exact nature of the disease is as yet undetermined.

MYELITIS.

Myelitis is a diffuse inflammation of the spinal cord. It may be acute, subacute, or chronic. It may be limited to one or to two adjacent segments, and then it is called transverse myelitis. It may be more extensive and effect various levels, and then it is called disseminated myelitis. It may progress either upward or downward from its starting point, and then it is called ascending or descending myelitis. It results in a destruction of the spinal elements.

Myelomalacia is a condition of necrosis of the spinal cord due to embolism or thrombosis of the spinal arteries. It, too, results in a destruction of the spinal elements. The symptoms of the two affections so closely resemble each other that they may be studied together.

In former years myelitis was supposed to be a common disease and occupied much space in text-books. But as the spinal-cord affections, one after another, have been carefully differentiated, and as the knowledge of multiple neuritis in its various forms has grown, it has become evident that many cases formerly considered myelitis were actually of a different nature. My own experience leads me to regard myelitis as a rare affection.

Etiology. Myelitis is, in the majority of cases, an acute infectious disease. It may be the only manifestation of infection, although its bacillus still remains undiscovered. It is often secondary to some other infectious disease.

The site of this infection may be a cutaneous or mucous surface which is open to the admission of germs. Thus Auche and Hobbs, Marinesco and Oettinger, Roger and Damaschino, Westphal and Leyden, have seen cases of myelitis following smallpox; Strümpell has observed a case of myelitis following a felon; Hochhouse has published a case of myelitis following tonsillitis; Ebstein, Hochman, Laveran, Schiff, and others have observed a myelitis following typhoid fever; Roger and Damaschino, Laveran, Hoffman, Babes, and Happel have observed myelitis following dysentery; Gull, Leyden, Ullman, Raymond, and Hayman have seen cases following gonorrhœa; Geoffrey and Achard, Bettheim and Foerstner have seen cases following pneumonia; Laveran, Leyden, Evan, Babes, and Varnalli have seen cases following malaria and grippe; Hochhouse has published a case following cystitis and pyelonephritis; Oppenheim one following abscess of the antrum and one following endocarditis. All forms of meningitis, both the epidemic cerebro-spinal meningitis, tuberculous meningitis, and purulent meningitis, may be followed by myelitis, either localized or general; and after wounds of the cord or operations upon the cord that are not aseptic, access of microbes directly to the organ may cause myelitis. It is thought that occasionally the access of germs to the cord may be along the track of nerve trunks when a purulent neuritis is followed by an ascending neuritis and myelitis. Cases of this kind are rare and require further investigation. I have never seen such a case. The poison of rabies may cause an infectious myelitis.

Cases are upon record in which myelitis of an acute type has developed subsequently to an exposure to cold. Thus Schultze has described a case of a young man, otherwise in perfect health, who developed an acute myelitis after travelling in a very cold railway car for several hours, and another case developing in a young man who hunted upon a very cold day for several hours. As already stated elsewhere, it is probable that such exposure to cold results in a lowered resisting power of the organs to the invasion of bacteria already present within the body and capable of setting up an inflammatory process in any organ whose circulatory condition is materially altered. The same lowering of vitality may account for the successful attack by the bacteria upon the spinal cord after blows, injuries, or falls.

The class of the community most often exposed to the disease is the class of active workers—porters, longshoremen, those who are accustomed to lifting heavy weights or doing heavy work, and those who are upon their feet a great deal—also soldiers who are subject to long marches. Overexertion is an admitted cause. A great strain may be followed either by hemorrhage or by acute myelitis. Thus in a case of my own the effort made by a porter in lifting a heavy box resulted in a sudden attack of transverse myelitis attended by hemorrhage, as the autopsy demonstrated. Falls and blows upon the back are said to be responsible for some cases of myelitis. When these produce hemorrhage the symptoms resulting may be those of a transverse or disseminated lesion, but an acute inflammatory process is rarely set up by this cause. A low grade of chronic myelitis is known to follow railway injuries and other severe injuries of the spine, but cases of an acute inflammatory myelitis have not been observed. A certain number of cases of acute myelitis develop after confinement. Whether these are due to infection by way of the uterus, or whether they are due to anæmia of the cord consequent upon hemorrhage, or whether they are due to a slowly acting venous congestion due to general enfeeblement, has not been determined. Syphilis is a common cause of acute myelitis. If the cord is not affected by gummy exudations in the membranes or syphilitic deposits within the cord itself, syphilitic changes in the bloodvessels with obliterating endarteritis and consequent thrombosis of the finer vessels of the cord is the method by which the disease attacks this organ. The form of disease described by Erb as syphilitic spastic paralysis has been found to be a chronic myelomalacia of the lower dorsal region due to endarteritis with secondary degeneration.[1] In some forms of poisoning, notably lead, mercury, phosphorus, and occasionally alcohol, disseminated foci of myelitis have been discovered, as well as multiple neuritis, and this has led to the supposition that the cord may be directly affected by toxins and toxic agents. That certain agents have a selective action upon the spinal cord is evident from our knowledge of the action of strychnine, which excites its activity, and of the coal-tar products and bromide and chloral, which diminish its activity. It is not impossible that the general feebleness and tremor occasionally seen in chronic bromide poisoning is due to defective nutrition of the spinal cord.

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[1] Lancet, October, 1902.

Males are affected more frequently than females by acute myelitis and by myelomalacia. This is because they are more commonly exposed to the usual causes of the affection. While no age is exempt, the majority of the cases occur between the ages of twenty-five and forty-five years.

Pathology. The pathological changes in acute myelitis have been best described by Marinesco.[1] The location of the lesion may be limited to one or two segments of the cord, under which circumstances one of the symptomatic types of transverse myelitis is produced, or the lesion may be disseminated through the various segments of the cord, normal tissue intervening between the inflammatory foci, when the clinical aspect is that of disseminated myelitis. The lesion may be diffuse with a tendency to extend from one segment to another, either upward or downward, or in both directions simultaneously, in which case the clinical picture is that of general myelitis or of an ascending or descending myelitis.

The gross appearance of the cord is not altered in these conditions, though palpation may reveal certain portions opposite the lesion which are softened to the touch. On cutting the cord across at these softened regions it swells up at the point of section, presenting a semifluid, creamy consistency, all differentiation between gray and white matter being obliterated, and the mass being either white and yellow from the admixture of pus, or red from the admixture of blood. When the disintegration has not been so extreme the cross-section shows an irregularity of the demarcation between gray and white matter, points of congestion, and irregular plaques of yellowish or reddish color, as evidence of infiltration of the cord with inflammatory corpuscles. In other cases plaques here and there of a whitish-yellow color indicate the formation of connective tissue as the result of a process which has fully run its course. The microscopic appearance of a section of the cord in the lesion of myelitis is shown in Figs. 121 to 124.

The vessels of the cord are enormously congested everywhere, and around the vessel walls emigrated leucocytes and small nuclei or round cells are present. Bacteria are to be seen by appropriate staining, many lying loose in the tissue, having come from the blood-vessels; others incased in leucocytes. The intense congestion is attended by rupture of the vessels, and small hemorrhages are evident throughout the inflamed tissue. Such hemorrhages are probably the result of destructive changes in the vessel walls produced by the bacteria. The infiltration of the cord by leucocytes is most extensive. (Fig. 122.) They collect particularly about the cells of the anterior horns of the cord. (Fig. 123.) The cells of the cord themselves are swollen and undergo various forms of degeneration with or without vacuolization, and general disintegration and

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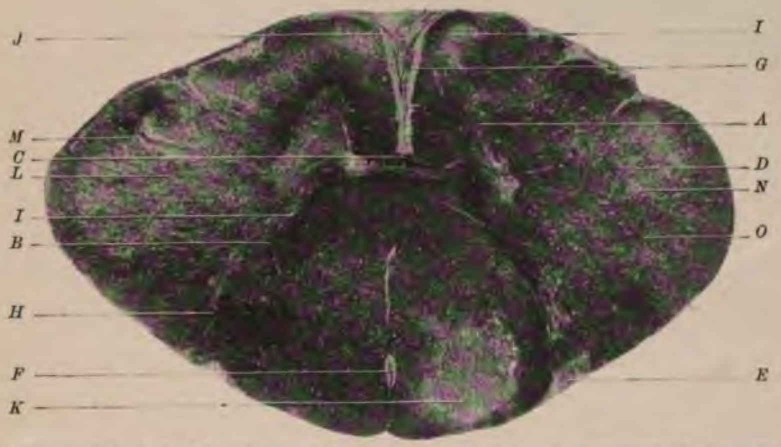
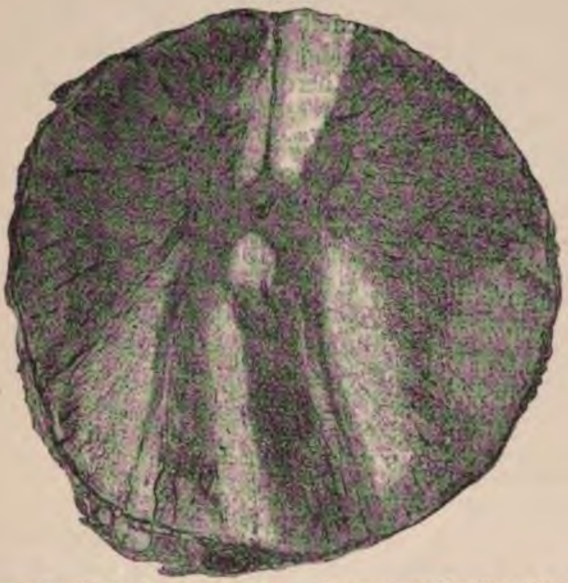
[1] International Congress of Medicine, Paris, 1900.

FIG. 121.



Acute myelitis at first dorsal segment. The inflammation is diffuse throughout the section, the lighter parts being most seriously affected. The distention of the bloodvessels is evident. *A*. Anterior horn. *B*. Posterior horn. *C*. Anterior commissure. *D*. Posterior commissure. *E*. Posterior nerve root. *F*. Posterior septum. *G*. Anterior fissure. *H*. Column of Burdach (normal). *I*, *J*, *L*. Inflamed areas in anterior lateral columns. *K*. Inflamed area in posterior column. *M*. Longitudinal section of thickened artery. *N*. Transverse section of thickened artery. *O*. Lateral column slightly inflamed. (Blocq.)

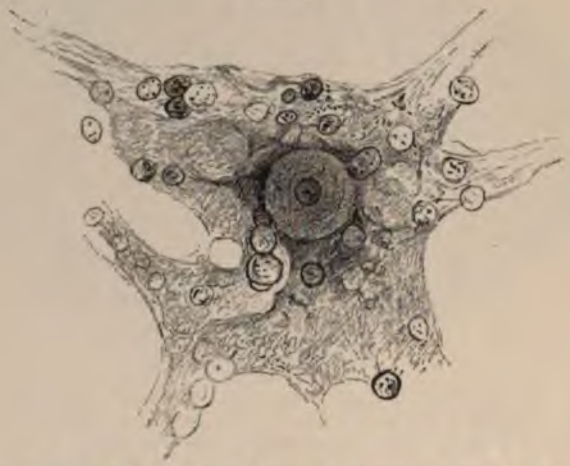
FIG. 122.



Acute transverse myelitis in dorsal region, due to the presence of bacillus coli and streptococci. The white columns are infiltrated and in a state of acute inflammation. (Marinesco, International Medical Congress, Paris, 1900.)

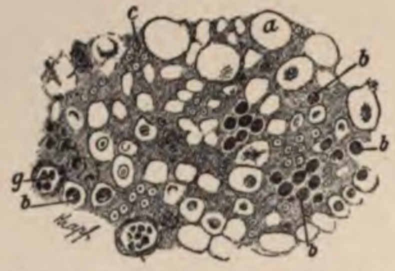
destruction both of the cell body and of the dendrites and axones. (Plate XV.) The nerve fibres are everywhere compressed by the products of exudation, and such compression results in a degeneration. The myelin sheath of the nerve fibre may be directly attacked by the leucocytes and disintegrate, forming fatty globules. The axis cylinder is very frequently swollen to several times its normal diameter by the imbibition of fluids. As a result of this long-continued tumefaction, there is a distention of the interstitial framework, and when the cylinders are subsequently disintegrated, degenerated, and absorbed, this enlarged sheath is left as a cavity, giving the cribriform or Swiss cheese-like appearance to the cross-section of the cord. (Fig. 124.)

FIG. 123.



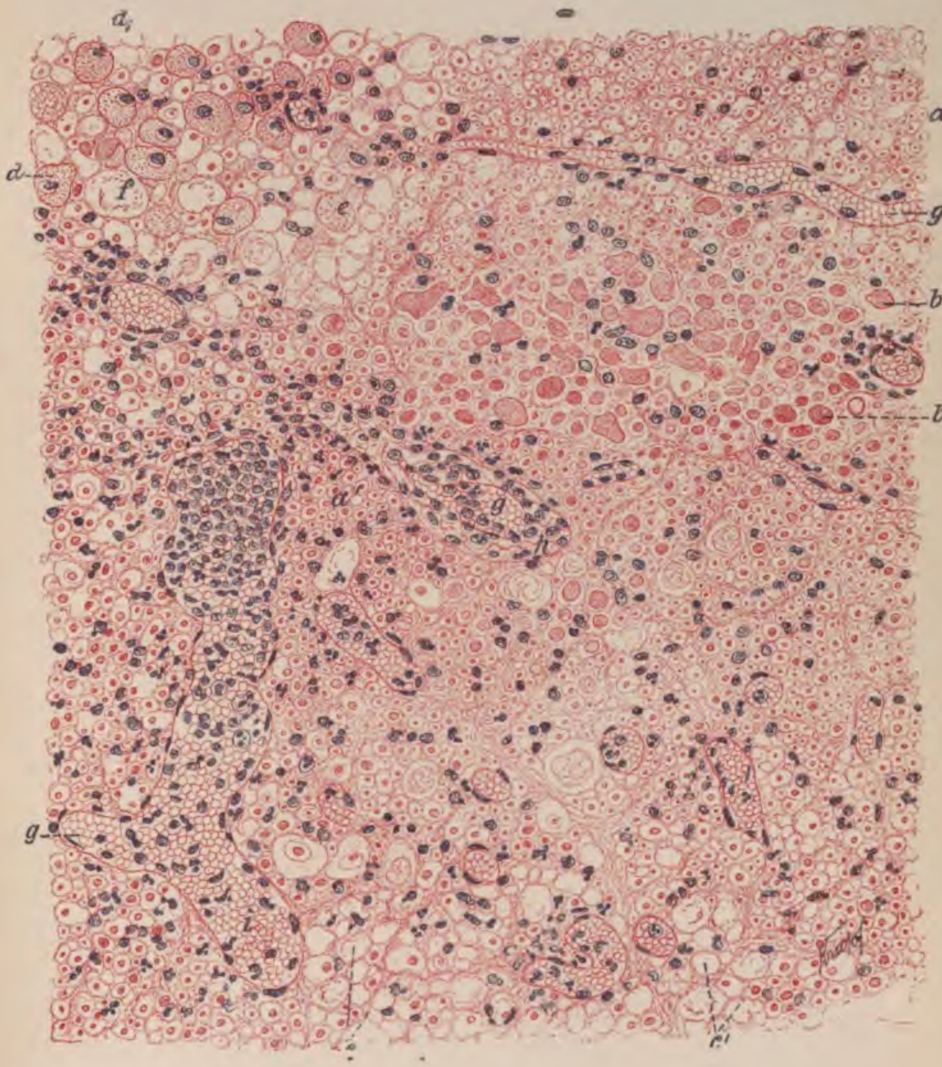
Motor neurone body of spinal cord from a case of experimental myelitis produced by streptococci. The cell is invaded by leucocytes, within which the streptococci are visible. (Marinesco, International Medical Congress, Paris, 1900.)

FIG. 124.



Acute myelitis. Cribriform appearance, due to distention of the sheath and subsequent absorption of the axones. *a.* Empty space. *b.* Swollen axis cylinders. *c.* Normal nerve fibre. *g.* Vessels. (Schmaus.)

PLATE XV.



Acute Myelitis, Infiltrating Form. (Schmaus.)

a, a_1. Less altered tissue. *b*, b_1. Swollen fibres. *c*, c_1. Distended spaces in the glia, with loss of fibres. *d*, d_1. Granular cells with their fat removed. *f*. Remains of destroyed nerve fibre. *g*. Vessel with many red and white cells. To the left of the picture the tissue is infiltrated with cells, especially in the lymph sheaths.

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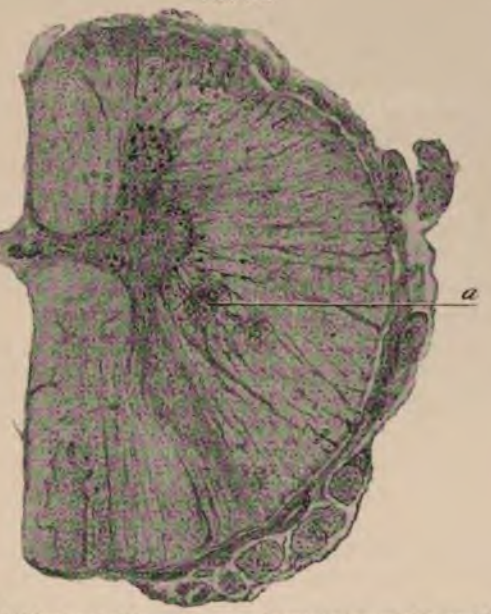
At the same time there is a very great change in the neuroglia cells. They become swollen, are increased in number, show karyokinetic figures, and produce a new structure of neuroglia within the spinal cord. (Fig. 125.) Such nodules of neuroglia are frequently crowded with microbes. Marinesco considers that this reaction of the neuroglia cells is primary and produced by the irritation of the microbes. It thus differs from the secondary sclerosis that follows upon the destruction of the nerve cells and fibres, and which is a replacement hyperplasia. The change in the nerve cell following the swelling of the cell is a pallor of its chromatophile elements and a chromatolysis with disintegration of the achromatic substance and gradual mutilation of the cell body. Granular bodies as well as very numerous fine nuclear cells are found infiltrated throughout the gray and white matter. Many of these cells are supposed to be phagocytes in process of destroying the myelin and the axis cylinders. The tendency of these phagocytes to invade and destroy both neurones and fibres of the nervous tissue has led Marinesco to name them "neuronophages." He believes that many of them come from proliferated neuroglia cells. Various bacteria which have been found in the spinal cord, producing this infectious myelitis, are the streptococcus, the white staphylococcus, the yellow staphylococcus, the pneumococcus, and a special diplococcus.

FIG. 125.



Meningomyelitis of infectious origin. *a*. Focus of acute inflammation and infiltration by new cells. The margin of the cord and the column of Goll are also in a state of infiltration. (Marinesco, International Medical Congress, Paris, 1900.)

The result of such a disintegration and inflammation of the spinal cord may be its total destruction, changing it into a softened mass, purulent in character. This is the condition in the rapidly fatal cases. In other cases the destructive process is less complete, the inflammatory conditions gradually subside, and there is left a mass of scar tissue, plaques of neuroglia replacing the nerve elements that have been destroyed. Any true regeneration of these nervous elements in the spinal cord appears to be impossible when the inflammation has gone beyond a certain point. It is true that the cells if not seriously damaged may resume their function, but when nerve fibres within the spinal cord have been disintegrated no regeneration appears to be possible, and hence permanent damage to the cord is always the result of an acute myelitis. A complicating meningitis is not infrequently found in infectious myelitis.

There are many cases of acute myelitis in which careful investigation fails to reveal the presence of microbes. It is possible, first, that the microbes have been present and have set up an inflammatory process but have subsequently disappeared from the foci of inflammation, a condition which is known to occur in other organs; or, secondly, that the inflammatory process is set up by toxic agents in the blood or toxins and not by the direct presence of bacteria. That the spinal cord can be attacked by toxins is evident from the changes that have been demonstrated in it in cases of alcohol, arsenic, and lead poisoning (see page 118) following diphtheria, after poisoning by ergot, by lathyrus, and by strychnine. Similar changes without bacterial influence result from trauma and are described in the chapter upon Injuries of the Spinal Cord.

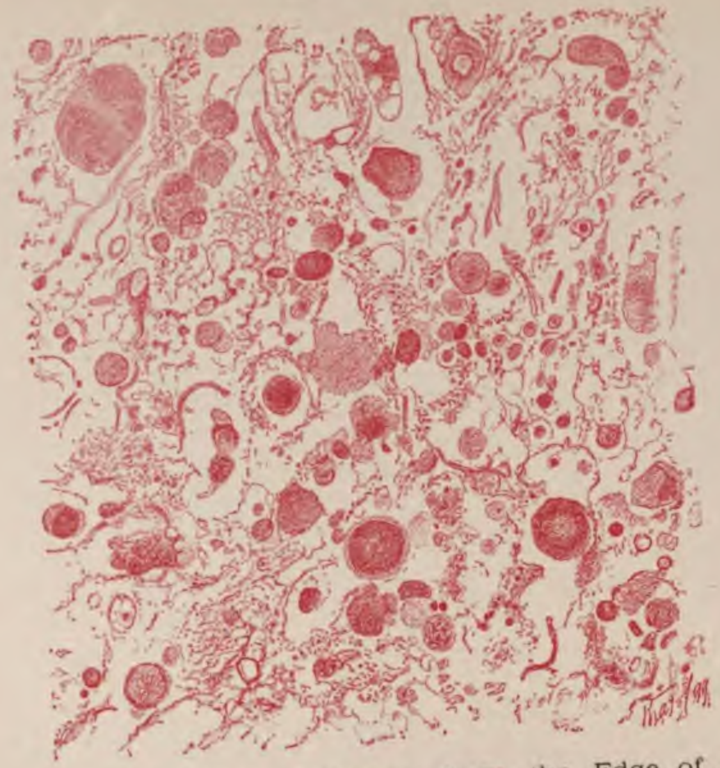
MYELOMALACIA.

Myelomalacia or softening of the spinal cord is a lesion resulting from embolism or thrombosis in the arteries or veins of the spinal cord. Such a softening may be red when accompanied by a transudation of blood cells or by minute hemorrhages, or white when the condition has been one of simple death of the tissue, or it may be yellow if the process is not observed until after a fatty degeneration of the débris and of the neuroglia elements has ensued. Microscopic examination shows a mass of débris, cells, and fibres in a state of disintegration, balls of myelin, swollen or small granular bits of axones, drops of fat, and red blood globules.

In all of these conditions the neuroglia presents a state of hyperplasia with nuclear formations which may be either primary or secondary. If the patient lives and the products of disintegration are absorbed their place is taken by new connective tissue, leaving in the place of the nervous tissue masses or patches of sclerosis. The initial lesion, as well as the sclerotic patch which is its result, is irregular in outline and very variable in extent, the conditions

PLATE XVI.

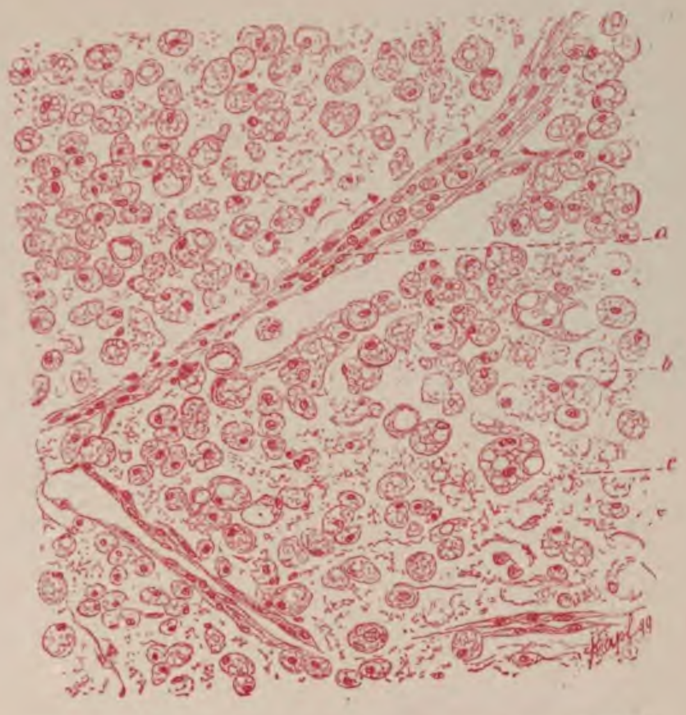
FIG. A.



Beginning White Softening from the Edge of a Recent Focus. (Schmaus.)

In place of nerve fibres there are round and irregular swollen masses, the glia is loose and torn, and its meshes are large. × 250.

FIG. B.



Section from a Focus of Softening, about Fourteen Days Old. (Schmaus.)

a. Connective tissue persisting. b. Granular bodies minus their fat. c. Detritus. × 250.

being such as to prevent any strict limitation of the softening in any direction, as has been particularly shown by Obersteiner and Redlich.[1] The result of a diminution in the blood supply of the spinal cord, of a suspension of nutrition consequent either upon ischæmia, or upon inflammatory conditions, is a parenchymatous degeneration of the neurones and of their branches in the region affected; hence the changes that are characteristic of myelomalacia may be present in the vicinity of foci of acute myelitis. In these conditions the nerve cells are much swollen and disintegrated, the nerve fibres are very much swollen, producing distention of their sheaths and the cribriform appearance in the white matter of the spinal cord already described. This condition should be considered as a necrosis rather than as an acute inflammation, but its result is the production of scar tissue of the nature of neuroglia, and hence the terminal state cannot be differentiated from the terminal state produced by acute myelitis. Sometimes, however, when such a scar is distinctly wedge shaped with base toward the periphery it will indicate that the origin of the trouble was an obstruction in a vessel entering the cord from the surface. Marger[2] has demonstrated that out of seven cases of supposed acute myelitis six were cases of pure necrosis, and Douglas Singer[3] has found this to have been the condition in seventeen out of nineteen cases examined by him. The chief pathological distinction between this form and the one already described consists in the absence of any emigration of leucocytes about the bloodvessels.

In many cases changes in the walls of the bloodvessels, thickening of the intima, or an infiltration of the adventitia, or a true endarteritis obliterans, with diminution of the lumen of the vessel and the formation of thrombi within it, have been observed. Marger believes that the spinal cord is particularly liable to the formation of necrobiotic spots on account of the peculiarity of its circulation and the defective anastomosis between the branches of the bloodvessels entering by the anterior fissure and those entering from the periphery of the cord. It would seem as erroneous to call these changes of myelomalacia myelitis as it would to name the corresponding changes that occur in the brain after embolic and thrombotic softening cerebritis. In the disease clinically described as myelitis we therefore include cases of myelomalacia or pure softening of the spinal tissue, which, when they do not cause death, go on to the formation of sclerotic patches in the spinal cord. (Plate XVI.)

Inasmuch as the spinal cord does not appear to be particularly susceptible to attacks of bacteria, as is shown by the rarity of myelitis as a complication of the infectious diseases, it seems probable that the majority of the cases of so-called acute myelitis are really of vascular origin. Whether a clinical distinction can be reached between these two types on account of the development in the infectious type of a marked febrile invasion with symptoms of an acute infection and leucocytosis, which are wholly absent in the second type, is a matter for further investigation. But the facts already cited in regard to two separate and distinct modes of invasion in poliomyelitis would point to the possibility of a similar distinction in cases of acute myelitis. And the much greater frequency of lesions in the brain depending upon vascular changes and vascular disease gives certain grounds for the hypothesis that lesions of the spinal cord hitherto thought to be inflammatory in nature are actually secondary to vascular disease. This fact, first pointed out with much force by R. T. Williamson,[1] has been substantiated by many subsequent writers, and is now generally accepted.

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[1] Handbuch der prakt. Med. Ebstein and Schwalbe, 1900.
[2] Ueber Myelitis Acuta, Obersteiner's Arbeiten, 1900.
[3] Douglas Singer. Brain, 1902.

The so-called spinal spastic paralysis of syphilitic origin described by Erb and known as spinal syphilitic paraplegia is really a myelomalacia of the mid-dorsal region of the cord due to endarteritis obliterans of syphilitic origin and followed by secondary degenerations. This is admitted by Erb in a recent review of the subject[2] from a study of the pathology of a number of cases.

Secondary Degenerations. When, in transverse myelitis or in softening or injury of the spinal cord, destruction of the nerve fibres passing through the segment affected occurs, these fibres are necessarily cut off from their connection with their cell bodies. And inasmuch as such a severance of connection is always followed by a degeneration from the point of injury to the peripheral termination of the fibre, it follows that, as a result of a limited transverse lesion in the cord, extensive secondary degenerations are produced. These degenerations begin within twenty-four hours of the original lesion, and go on progressively for six or eight weeks until the nerve fibres are completely broken down. As a result of this degeneration both the axone and the myelin about it are disintegrated and finally liquefied and absorbed. Hand-in-hand with this progressive degeneration of the fibres there occurs a compensatory thickening of the neuroglia, forming a framework about the fibres, and hence as the terminal lesion of such secondary degenerations we have sclerotic conditions in the cord, of greater or less extent, according to the greater or less extent of the fibres originally degenerated. (See Plate XVII.)

Below the lesion such degeneration is termed descending degeneration, because it affects the fibres whose neurone bodies lie at or above the transverse lesion. Such descending degeneration is very extensive near to the lesion, inasmuch as there are descending fibres in all the various columns of the spinal cord. (Plate XVII., D. VIII.) At a distance of several segments below the lesion the descending degeneration is more closely limited to the long-descending columns of the cord. It is then manifest chiefly in the anterior median and in the lateral pyramidal tracts which contain the long motor fibres from

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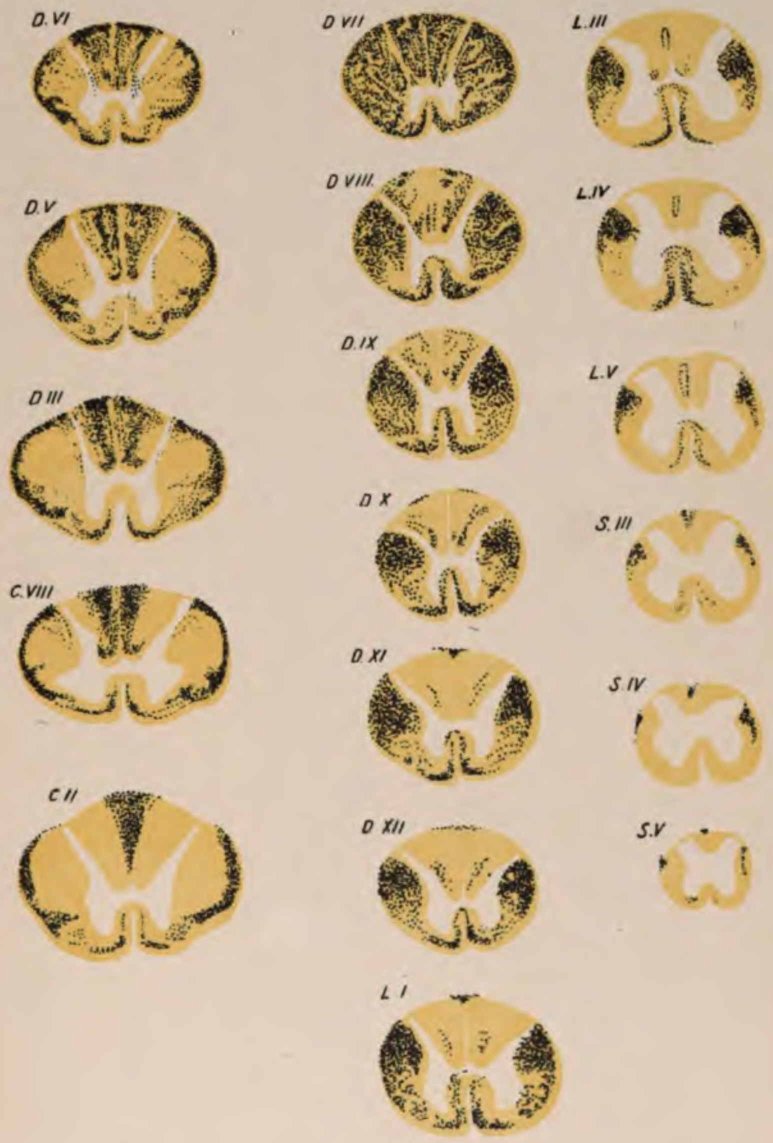
[1] Manchester Medical Chronicle, 1895.

[2] Lancet, October 13, 1902.

PLATE XVII.



Ascending (D. VI to C. II) and descending (D. VIII to S. V) degeneration in the spinal cord after a transverse lesion at D. VII. Marchi stain.

(Hoche, Arch. f. Psych., vol. xxviii., plate ix.)

the brain to the anterior horns of the cord. (Plate XVII., D. XI.) There are descending degenerations also evident for several segments in the anterior and antero-lateral columns, these degenerating fibres being in the association tracts of the cord. (See page 186.) In the columns of Burdach there is also a small area of degeneration, the comma-shaped column of Schultze, which degenerates downward for three or four segments. (Plate XVII., D. X.) This column contains fibres of the posterior nerve roots which turn downward after their entrance into the cord and terminate in the gray matter of the third or fourth segment below their entrance. In the column of Goll, adjacent to the commissure lie a few fibres which degenerate downward for a short distance after a transverse lesion. If the transverse lesion is in the lumbar or upper sacral region there is a descending degeneration in the septomarginal tract—oval field of Flechsig. (Plate XVII., S. III.)

Ascending degeneration after a transverse lesion is found in the anterior and antero-lateral columns of the cord for a short distance, the association fibres whose neurone bodies lie in the parts below the lesion degenerating upward. The column of Burdach is very considerably degenerated above the level of the lesion, and the fibres of the posterior nerves, which come into the damaged segment, degenerate upward together with other fibres that have entered the cord below the level of the lesion, but have not yet terminated in the posterior horns. But as normal posterior nerve roots enter the column of Burdach at levels above the lesion, the area of ascending degeneration in this column becomes more and more limited as higher levels are examined. The column of Goll degenerates upward in its entire length after a transverse lesion of the cord, but the extent of this degeneration in any single segment will depend upon the level of the transverse lesion. It will be remembered that the column of Goll contains all the fibres from the sacral, lumbar, and mid-dorsal segments of the cord that pass upward to the medulla. The higher the level of the lesion in the cord the greater the extent of the degeneration in the column of Goll. Figs. 47 and 48 show the varying extent of secondary degenerations after transverse lesions at different levels. The direct cerebellar column degenerates upward after a transverse lesion of the cord. Such ascending degeneration is not evident, however, if the lesion lies in the lumbar or sacral region, inasmuch as this column begins at the first lumbar segment. The higher the level of the lesion the greater the area of degeneration in this column. The column of Gowers (antero-lateral ascending tract) degenerates upward in its entire length after a transverse lesion of the cord, and the area of this degeneration is greater the higher the level of the transverse lesion. These degenerations are well shown by the Marchi stain in Plate XVII.

Secondary degeneration occurs after compression or lesion of the cord at a single segment from whatever cause; thus, the compression of the cord by tumors, or by caries of the spinal column, or injuries of the cord by fractures of the spine, or by hemorrhages outside or within the cord, or by wounds of the cord are followed by these secondary degenerations.

Symptoms. The symptoms occurring in myelitis and myelomalacia depend chiefly upon the location of the lesion. Two kinds of symptoms develop which can be sharply differentiated from one another. The first class of symptoms may be termed direct symptoms, and are due to destruction of the elements of the spinal cord at the point of lesion. These consist of (*a*) paralysis, atrophy, and reaction of degeneration in the muscles which are supplied by the cells of the anterior horns destroyed; (*b*) loss of the reflex action controlled by the segment which is affected; (*c*) paræsthesia and anæsthesia in the area of skin related to the segment which is destroyed. The region of anæsthesia, as outlined upon the body of the patient when compared with Plate XIII., page **193**, is the best guide to the exact level of the lesion. There may be a condition of hyperæsthesia just above the level of anæsthesia corresponding to the two segments of the cord just above the level of the lesion. There is usually pain, and tenderness to pressure in the back. Hot applications are painful opposite the segment of the cord which is invaded. This is due to the hypersensitive condition of the segments just above the lesion.

The second class of symptoms may be termed indirect symptoms, and are produced by the cutting off of impulses to and from the parts of the cord below the point of injury. It will be remembered that the cord transmits many impulses upward and downward to and from the brain. In transverse myelitis the tracts conveying these impulses are damaged; hence a certain number of symptoms develop, due to interruption of the motor tracts to the parts below the lesion and to interruption of the sensory tracts from the parts below the lesion. These indirect symptoms are associated with the secondary degenerations already mentioned as a result of the lesion. Such indirect symptoms are (*a*) motor, consisting of paralysis of voluntary motion in the legs with an increase of muscular tone in the paralyzed muscles. These become rigid, have a tendency to contraction, and produce spasms and contractures, with deformities in the joints. Such paralyzed muscles are not atrophied and have no reaction of degeneration; (*b*) the reflexes are markedly increased in the parts below the lesion, so that an exaggerated knee-jerk, a crossed adductor reflex (tapping on the knee of one side produces adduction of the opposite thigh), ankle clonus, and Babinski's reflex are present; (*c*) there is a loss of the control of the bladder and rectum, attended by retention of urine and involuntary evacuations of the bowel; (*d*) sensations in the parts below the level of the lesion are uniformly impaired in all qualities: touch, temperature, pain, and the muscular sense being affected; (*e*) trophic symptoms, especially bedsores over the sacrum, trochanters, and heels and ankles. These develop early, on account of venous congestion in the skin, intensified by too long-continued pressure, the position not being changed and the skin being irritated by fine particles of dirt or infected through small abrasions; they often become extensive and deep, secrete an ichorous, offensive discharge, expose the bone, which becomes necrotic, and cause a general septic condition which may be fatal; cystitis is usually to be ascribed to infection from dirty catheters or by an ascending urethritis, and is rarely, if ever, a trophic lesion of the bladder; (*f*) vasomotor disturbances, consisting of blueness and coldness of the skin, distention of the veins, and increase of sweat. The sweat may be offensive.

The onset of these symptoms may be sudden, or they may develop gradually in the course of a week or ten days. In the acute cases, which come on within a few hours, the probable condition is one of myelomalacia or a primary vascular lesion. In the subacute cases, which develop more slowly, an infectious or inflammatory origin is more likely. The occurrence of a rise of temperature and marked febrile symptoms point to the latter condition.

The course of the case is a slow one, and the gradual recovery from the severe paralysis to the state of partial incapacity takes several months or years. The patients are said to be in a state of chronic myelitis during their life of invalidism. Occasionally in the lighter cases complete recovery ensues. But the damage to the cord, if serious, cannot be repaired, and hence no complete or permanent recovery is possible. On the other hand, if the causes of death, which are cystitis, inflammation of the rectum, sepsis from bed-sores, and pneumonia, can be avoided by care and attention to the patient, the prospect of recovery from the attack is good. A permanent condition of chronic myelitis, the result of secondary degenerations, remains for the rest of the patient's life, and this is manifest by a greater or less degree of spastic paraplegia in which the limbs are stiff, the knees overlap in walking, there is trepidation of the feet when the weight rests on the ball of the foot; there are spasmodic contractions and cramps in the legs, there is more or less loss of sensibility in the limbs, and an inactivity of the bladder and rectum, so that the catheter has to be used regularly and the rectum washed out regularly. Hence a state of invalidism remains, from which, however, the patients do not die unless some complication arises. The chief danger to which they are subject is an infection by the catheter, consequent cystitis, pyelonephritis, and uræmia.

These various symptoms of myelitis occur in varying combinations. The most common type is transverse dorsal myelitis, of which the following case is a fair example and demonstrates the course of the disease as well as the various symptoms which appear.

Male, aged fifty-six years, of good habits, not syphilitic and perfectly healthy, after a long walk perceived sensations of numbness and tingling in the feet and legs as high as the knees. Instead of resting, he attempted by further walking to wear off this numbness, but before he had proceeded one-half a mile was conscious of great weakness in the feet and legs, and within one-half hour was considerably paralyzed, so as to be unable to stand. The paralysis was total in the legs below the knees. It was impossible to pull the knees up in bed, though they could be extended and slightly adducted. Within two hours all sensation was lost up to the level of an inch below the umbilicus, and at this point a distinct girdle sensation was perceived. Examination showed loss of sense of touch, temperature, and pain up to this level, and for two inches above this level a condition of extreme hyperæsthesia to touch, pain, and temperature. The muscular sense was not lost in the legs, and all changes of position in the feet and toes was acutely perceived. Retention of urine was present, and there were no sensations in the rectum and no control over its action. There were no symptoms above the level of the umbilicus. There was no fever. This condition remained stationary for about four months, during which time, by care in the emptying of the bladder and of the rectum, and by frequent change in the position of the patient in bed, complicating cystitis, proctitis, and bed-sores, which were constantly threatened, were avoided. The patient then began to recover slightly the power in the legs, but was much troubled by twitching of the muscles and sudden spasmodic contractions of the legs, sometimes in the form of flexion of the knees, sometimes in the form of extension of the foot. The paralyzed muscles did not atrophy, and the electrical reaction remained normal. Knee-jerks, which were completely lost during the first week, then returned and gradually became exaggerated, and after two weeks ankle clonus, Babinski's reflex, and crossed reflex of the knees appeared and have continued. A gradual progressive recovery of power and of sensation ensued during the following two years, but was attended by an increasing rigidity of the legs. His sexual power returned. A stationary condition has remained for four years in which the patient has a spastic rigidity of both legs, and, though able to walk, does so with stiffness and great effort with the aid of two canes, there being tendency of the knees to overlap, a dragging of the feet, and a frequent trepidation of the foot when the weight happens to rest upon the ball of the toe. It is impossible for him to rise from the chair or to sit down without assistance. There has never been a return of the control of the bladder and rectum, there being retention of urine, which is relieved by the catheter. Sensations have returned in the previously anæsthetic area, but are by no means as keen as in the hands and arms. The girdle sensation remained for two years and then subsided. There is no longer a zone of hyperæsthesia.

It is evident from this case that there was an acute onset, then a stationary period lasting several months, and then a gradual improvement, which progressed for six years, leaving the man at the end of that time in a stationary condition from which no recovery is likely. The level of the lesion was the tenth dorsal segment, as was shown by the level of the anæsthesia. Inasmuch as the functions of the tenth dorsal segment of the cord are very few, the direct symptoms in this case were quite subordinate to the indirect symptoms due to a cutting off of the tracts passing through this segment.

I have seen a case in which a rapidly advancing transverse myelitis at the sixth dorsal segment left the patient totally paralyzed and absolutely anæsthetic below this level. This condition had been present four years when I saw him, and during all this time bed-sores had been prevented by having him turned in bed every half-hour day and night. The pressure of his body weight caused a redness, followed by cyanosis in the skin, which would surely have gone on to a bed-sore had this change of posture not been made. In this patient, although all sensation was abolished below the level of the eighth intercostal nerve, any irritation of the skin in the anæsthetic region produced a sensation which was referred to the skin in the hyperæsthetic band at the level of the sixth intercostal nerve. Such erroneous location of sensations I have observed in several cases of myelitis, but have not seen it described.

Transverse lumbar myelitis is the next most common form of myelitis, and here the lesion may be limited to the lumbar segments or may involve both lumbar and sacral segments of the spinal cord. As an example, the following case may be cited:

Male, aged sixty years, of fairly good habits and without any ascertainable cause, was suddenly attacked by a tingling and numbness in the legs as high as the knees and in the gluteal region. This numbness was followed in the course of twenty-four hours by a condition of marked weakness in the feet and legs, with drop-foot and with retention of urine and inability to control the rectum. He had considerable dull pain in the back over the sacrum and down the back of the thighs and legs. Within four hours of the onset he was completely unable to move his legs in bed in any direction, though when the knees were pulled up slight adductor movements of the thighs could be produced voluntarily. An extensive area of anæsthesia was found in the legs and in the back and outer part of the thighs, but a funnel-shaped zone on the front of the thigh was found hypersensitive rather than anæsthetic, and there was no disturbance of sensation above the level of Poupart's ligament in front. The knee-jerks were lost, the muscles that were paralyzed in the legs were flaccid, soft, and soon began to atrophy, later on showing partial reaction of degeneration. He had a slight fever, the temperature varying from 99.5° to 101° F. for six days, when it became normal. At the end of two weeks all the symptoms began slowly to improve. In the course of three months he had begun to move his limbs, though by this time they were markedly atrophied. He had developed a cystitis due to imperfect catheterization, and was still unable to control the action of his rectum. The anæsthesia, though present, was markedly diminished in intensity, and there was no point upon the limbs where sensations of touch and pain could not be located. A subjective difference, however, between the sensations of touch, temperature, and pain, as felt in the legs and hands, was present. During the following three months the improvement gradually went on, and seven months after the onset he was able to walk with a cane, but was still unable to go up and down stairs. He could rise from a chair by the aid of his hands, but in walking there was a marked weakness of all the muscles and a tendency to drop-foot. Numbness was present over the legs and back of the thighs, and it was still necessary to use a catheter. This condition has persisted for the past five years, the legs being weak, thin, and somewhat insensitive. The knee-jerks have never returned, and the action of the bladder has still to be assisted. He is impotent. The entire lumbo-sacral enlargement up to the second lumbar segment was evidently diseased, but the lesion was nowhere so completely destructive as to suspend motor and sensory functions.

I have seen a similar case, developing after a confinement that had been attended by profuse hemorrhage but no apparent sepsis. This patient went on slowly to complete recovery in six months.

When the cervical region of the cord is invaded by transverse myelitis the direct symptoms appear in the arms and hands, the hands and arms below the elbow being affected if the lesion is in the lower part of the cervical region, and the arms and shoulders if the lesion is as high as the fifth cervical segment. A rapid onset of total paralysis with anæsthesia and reaction of degeneration in the muscles affected and loss of their reflex activity occurs, the anæsthesia on the body, however, being often lower down than might be expected in a cervical lesion of the cord. Thus in a case of sudden onset of paralysis affecting both hands and muscles of the forearms, excepting the supinator longus, and attended by anæsthesia in the inner surface of the arm and in the greater part of the forearm, excepting the radial side, the anæsthesia upon the trunk, together with the zone of hyperæsthesia above it, was below the level of the nipple. Paralysis of the sympathetic in the neck, causing a narrowing of the palpebral fissure, retraction of the eyeball and a loss of dilatation of the pupil on irritation of the neck were present.

The indirect symptoms of spastic paralysis in the legs develop early. The knee-jerks are exaggerated, ankle clonus appears, the legs are stiff and rigid, and subject to quick sharp spasms of a painful character, and cramps. There is usually retention of urine, though occasionally the bladder may empty itself suddenly without the control of the patient. There is no control of the rectum, which may act in the same manner as the bladder when full. In these cases it is not uncommon for priapism to occur. These cases are more dangerous than those of transverse myelitis of the dorsal and lumbar region, because of the possibility of an ascending myelitis which may invade the cells governing the phrenic nerve and cause death by respiratory paralysis.

In disseminated myelitis the symptoms of paralysis with atrophy and disturbances of sensation are more extensive than in cases of transverse lesion, and symptoms appear both in the legs, body, and arms simultaneously or in rapid succession. A few muscles in each limb may be picked out by the paralysis, adjacent muscles being weak, but showing no tendency to atrophy or reaction of degeneration. A disturbance of sensibility is usually marked, but does not go on to a total anæsthesia. There is commonly a loss of bladder and rectal control. I have never seen a case of disseminated myelitis, but in the following history by Taylor[1] there is a fair illustration of the course of the case:

A man, aged thirty-two years, slipped while getting off a train and received a slight shock, but did not fall to the ground. His body was bent backward, and he stepped forward suddenly in order to preserve his balance. At this time he felt a little pain in his loins and soon after pains throughout his body. He continued his work for the two following days. On the third day, after a long walk and an exposure to wet, he found himself unable to hold his water. On the fourth day he felt weakness in the legs and had difficulty in walking home. A day later there was marked weakness of the legs, retention of urine, and considerable numbness in the legs, and the day after he was confined to his bed with paralysis. On the ninth day after the injury he had involuntary movements and a temperature of 102° and 103° F. He was then taken to the hospital, and on the twelfth day after the injury was found to be completely paralyzed in the lower extremities, was able to move only the head and hands, the hands being weak. The lower part of his chest was immovable and the breathing was diaphragmatic. His voice was clear, but he had a short, weak cough, and could not expectorate. The abdomen was tense, tympanitic, not resistive. He had a complete loss of sensation up to the level of the seventh rib on both sides. There was a sensation of pins and needles in the upper extremities, but not in the lower. There was no reflex action in the legs and the muscles were not rigid. Electrical reactions were normal. Temperature continued high, 101.5° and 104° F. Three days later he had entire loss of power in the right hand, and the thoracic muscles were completely paralyzed. There were cystitis and febrile symptoms, and he was delirious. The following day his dyspnœa was excessive and he spoke with difficulty. He could still move his left hand slightly. He died of exhaustion. The autopsy showed an extensive softening of the spinal cord from a point three inches above the lower extremity to a point nine inches higher up, the softening being disseminated in small patches irregular in size.

Prognosis. In the early stage of myelitis a very guarded prognosis must be given. It is impossible to tell to what extent or severity the symptoms may develop, and until the progress is arrested the outlook is always serious. Infectious cases, especially those that are secondary, have a better chance of recovery than cases due to disease of the bloodvessels, and the slower the onset of the symptoms the better the chance of their arrest. The more absolute the anæsthesia in any case the worse is the prognosis. The greater the pain in the back, especially if it is attended by rigidity of the spine, or spasms in the legs, the worse the prognosis. After the acute onset the prognosis will depend on the severity of the symptoms. Bed-sores and cystitis make the outlook less hopeful. After very complete paralysis I have seen a slow but progressive improvement and a return of power to walk. It is always well to hold out the hope of some improvement for two years. After that time no change can be expected. The younger the patient the more vigorous his general condition, and the better his habits the better his chances.

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[1] Allbutt's System of Medicine, vol. vii, p. 12.

Treatment. Absolute rest in bed must be enforced from the outset. The patient should lie in a prone position, if possible, and great care should be taken to change the position at least every half-hour and to so distribute the weight, by means of pillows or by an air-bed or water-bed, as to prevent pressure of the body coming upon the bony points. Frequent sponging of the skin and careful attention to cleanliness, especially if there is an incontinence of urine or feces, are necessary in order to prevent the development of bed-sores. Under all circumstances the use of the bed-pan or urinal should be enforced, as the effort of sitting up may increase the symptoms. If the skin is very red it should be bathed with an antiseptic solution and with a solution of alum and covered with a powder, and no pressure on it permitted. If a bed-sore forms it is to be dressed like any ulcer and kept as aseptic as possible. Stimulants to healing, such as balsam of Peru or a galvanic current directly applied, have never seemed to me to be effective. The most important thing is to remove all pressure from the ulcerated surface, and thus allow it to heal. The greatest care should be taken in catheterization, all catheters should be kept in antiseptic solutions and carefully washed, and coated with carbolized vaseline before being used. It is too commonly the custom to limit the washing of the catheter to the time succeeding instead of preceding its use. If cystitis develops the bladder should be washed out twice or three times a day with a 10 per cent. solution of boric acid at 98° F., and the patient should be given salol, five grains, or urotropin, ten grains, every four hours by the mouth. The action of the bowels should be regulated by means of laxatives daily, which should be assisted by an injection given at a regular time of the day, a pad being constantly applied to the anus to prevent leakage. The spasmodic contractions and cramps in the muscles may be relieved by the use of warm clothes to the back or of warm bottles, care being taken not to burn a blister in the anæsthetic skin. Bromides are sometimes of much service for this symptom. Any active medicinal treatment for the disease during the period of onset or during the period of maximum intensity is usually futile, though salicylate of soda and salicin have been supposed to be of use in infectious cases. When the active process has come to a standstill and a period of improvement sets in, massage and electrical applications to the muscles, gradually increasing voluntary movements and exercises, tending to re-establish the voluntary control of the limbs, are to be used. Baths are also of service, either warm and cool sponging of the back, or warm douches under moderate pressure (ten pounds), or tepid sitz baths with sponging of the back. Cold (60° F.) or hot (100° F.) baths are to be avoided. When the patient begins to walk great care should be taken to prevent over-fatigue and to prevent falls. Rubber heels or soles help these paralytics. In some cases apparatus may be used to facilitate the act of walking, such as braces to the ankles or knees or a stiff corset to the back.

CAISSON DISEASE.

Etiology. Persons who have worked in caissons or in deep mines under high atmospheric pressure, or under the sea in divers' garments under high atmospheric pressure, are occasionally seized with paralysis on coming out into the ordinary air. This paralysis has, therefore, been ascribed to the sudden change of atmospheric pressure, it being supposed that the system accustoms itself to a very much increased pressure—say of four to six atmospheres—without difficulty, but the sudden removal of this is the cause of the paralysis. Hence, at the present time, where individuals have to work subjected to such pressures provision is usually made to remove this pressure very slowly. Thus modern mines and caissons have several intermediate chambers in which the workmen are advised to remain for one hour or more, thus accustoming themselves gradually to the ordinary atmosphere, and if this precaution is observed no ill results follow.

It has been found that ordinary healthy men of good habits are very much less liable to the disease than men who indulge in alcohol freely, or who have some form of heart or kidney disease, or who are very fleshy. Workmen gradually become accustomed to endure pressure, and hence there is a greater danger for new hands than for the older workmen.

Pathology. The pathology of the disease is not fully determined, though the lesions of myelitis have been found in all cases. It has been thought, from experimental investigations, that the development of gas in the bloodvessels has resulted in embolism of an extensive character in the capillaries, the emboli being bubbles of gas, and hence that the acute myelitis is a true myelomalacia from multiple emboli. In other cases where no such embolism has been discovered, multiple hemorrhages have demonstrated that a disturbance of the circulation, either of the nature of acute congestion with hemorrhage, or of the nature of acute thrombosis, has been present.

Symptoms. The symptoms of the disease develop very rapidly within a short time of the exit of the individual into the ordinary air. They are both cerebral and spinal symptoms. The cerebral symptoms consist of headache, prostration, feelings of faintness, nausea, vomiting and vertigo, double vision, and difficulties of speech and of breathing. Some patients vomit very freely and then become comatose. The spinal symptoms are more evident than the cerebral symptoms. Patients complain of severe pains which are usually felt in the legs and in the trunk, occasionally in the arms and back, and these pains persist even when all sensation is abolished. Pains are referred commonly to the joints, but may be referred to any part of the body, and in their intensity and severity resemble those of the first stage of tabes. They are attended by numbness and tingling or sensations of great cold or of heat. Soon after the onset of pain a feeling of weakness develops which goes on rapidly to a condition of paralysis, both legs being totally paralyzed and both arms being commonly affected soon after the legs. The sphincters are paralyzed, and the anæsthesia rapidly extends to all parts of the body. Variations in degree in all these symptoms have been observed, but a large majority of patients have been completely paralyzed within a few hours. In many cases the symptoms subside as rapidly as they have developed, and within a week the patient is able to be up and about. In other cases, however, all the symptoms of an acute myelitis ensue, and in the cases that have died examination of the spinal cord has demonstrated both transverse myelitis of the dorsal region of the cord with ascending and descending degenerations and also disseminated myelitis. Such patients go through the regular symptoms of acute myelitis, and either die or become chronic invalids.

Causes of death, as in acute myelitis, are usually cystitis, bedsores, or pneumonia.

Diagnosis. The diagnosis of the disease, therefore, rests upon the ascertaining whether the patient afflicted has been subjected to high atmospheric pressure and then has come back into the air very suddenly.

Treatment. A knowledge of the cause of this affection should lead employers to warn their workmen against coming immediately from a high to a low atmospheric pressure and should make it incumbent in all works to have a graduated series of chambers through which the laborers should be made to pass from a higher to a lower pressure. When the symptoms have developed it has been recommended to immediately return the patient to the caisson or to place him in a pneumatic cabinet where a high atmospheric pressure can be immediately produced, and then, by a gradual reduction of the pressure, to accustom him to the ordinary atmosphere. Such a pneumatic cabinet should always be accessible in works or mines where laborers are subject to this disease. When this is not feasible it has been recommended by those who have experience in this affection that large doses of ergot, one drachm every hour or every two hours, should be given. Bandaging the limbs tightly with an Esmarch bandage, and thus confining the blood to the larger cavities, has been employed with good effect. Later on the treatment should be that for acute myelitis.

CHRONIC MYELITIS.

Etiology. Chronic myelitis may develop as the result of an acute myelitis when this does not go on to a fatal termination but leaves a certain amount of damage to the spinal cord. A myelitis, however, may be from its onset a slow process, and thus the disease may be chronic from the start. Any of the causes capable of causing acute myelitis are equally capable of producing chronic myelitis. Disturbances in the circulation of the cord are probably the primary cause in the majority of cases, atheroma of the bloodvessels or syphilitic endarteritis being among the most common conditions found in this disease. Chronic myelitis may also develop as the result of chronic meningitis, and is usually associated with more or less implication of the spinal meninges. The term chronic myelitis may be applied to any or all of the processes of degeneration developing in the cord in connection with other spinal-cord affections. Thus the terminal stage of lateral and combined sclerosis, of amyotrophic lateral sclerosis, of acute or chronic poliomyelitis, or of disseminated sclerosis may be diagnosticated as chronic myelitis in case no exact history of the onset of the disease can be obtained. Chronic myelitis may also occur without any ascertainable cause, a chronic irregular degeneration, both in the white columns and in the gray matter being found after death in cases where the disease has been one of slow and irregular type. Chronic myelitis may develop in the course of very many constitutional diseases that produce disturbance of general nutrition, and it may also develop secondarily to many of the acute infectious diseases, being in these cases due rather to the toxic agents produced by the infection than to any direct bacterial action. Any of the various forms of injury of the spinal cord or of its bony covering, or of its meninges, may be followed by a chronic degeneration in the spinal cord. Very great and long-continued physical exercise is also supposed to be a cause. Chronic alcoholism may produce a chronic myelitis as well as a peripheral neuritis, and in some cases of very severe peripheral neuritis which failed to recover, changes have been found of a degenerative nature in the spinal cord. Thus chronic myelitis may be the result of a state of poisoning which produced first a multiple neuritis. Chronic myelitis develops occasionally as a sequel of gout with or without an attending multiple neuritis, the exact nature of the process being still a matter of uncertainty. Chronic myelitis is in some cases due to syphilis, and then it may be either a diffuse process advancing into the cord from the periphery, attended by degeneration which appears first in the lateral columns of the cord and gives rise to the symptoms of spastic paraplegia (the spinal syphilis of Erb), or it may be a transverse process due to syphilitic endarteritis in the vessels at a certain level, or to the appearance of syphilitic indurations with infiltration of round cells in both white and gray matter. Such syphilitic infiltrations may be very irregular in their distribution; may be confined to one segment of the cord, causing a chronic transverse myelitis; may be limited to one or more columns of the cord, causing symptoms resembling locomotor ataxia or lateral sclerosis of one or both sides; may cause a unilateral lesion, producing symptoms of Brown-Séquard paralysis, or may be irregularly disseminated throughout the cord. Chronic myelitis is sometimes due to pernicious anæmia, the changes in the cord which occur in the course of this disease being diffuse in location and slow in development[1] (see page 337). Slow poisoning by ergot or lathyrus may cause chronic myelitis.

Chronic myelitis is usually a disease of adult life, persons of the ages from twenty-five to forty-five years being more liable to the affection; it is about equally prevalent in males and females. It is possibly traceable in some cases to an inherited neuropathic tendency. It is, in my experience, a very rare form of affection, and it is probable that the descriptions found in the older text-books, which rarely correspond to cases now seen in the hospitals, were written before the time when careful distinction was made between the various forms of spinal-cord disease. As the knowledge of spinal-cord disease has increased it has been possible to separate accurately the different types, and the more this is done the rarer the diagnosis of simple chronic myelitis is made. In fact, some authors have denied that as a special disease it can occur, and affirm that it is always to be regarded as the terminal stage of some one of the diseases of the cord already considered.

Pathology. The pathology of chronic myelitis cannot be traced accurately from the onset, inasmuch as the terminal stage of the disease is the only one which is open to direct observation. In patients dying from chronic myelitis the spinal cord may present slight deformities in its contour, but the changes are only visible in microscopic section. These changes are of two varieties: first, the appearance of sclerotic patches irregularly distributed throughout the white and gray matter of the spinal cord. These sclerotic patches consist of a thickening of the glia and of the connective tissue about the bloodvessels, usually attended by a distention of the bloodvessels and by changes in their walls of the nature of endarteritis—both obliterating or atheromatous. As a rule, this sclerotic tissue is thicker and more dense than in the ordinary forms of secondary degeneration, or in tabes, or in lateral sclerosis. Occasionally in the mass of sclerotic tissue a distended axis cylinder has produced a cavity, and the cord presents the cribriform appearance, or Swiss cheese appearance, which has been described as occurring in acute myelitis. Usually the sclerosis is thicker at the periphery of the cord, and is associated with adhesions of the pia and with chronic meningitis. It has a tendency to be thicker around the bloodvessels that enter the cord from the periphery, and very often wedge-shaped patches of sclerosis are seen with the apex pointing inward. It is evident that the neuroglia of the cord is increased in density and also that the connective tissue in the vessel walls is thickened. All throughout this sclerotic tissue a fine cell infiltration is found, more intense about the bloodvessels, but everywhere present in the cord. If this irregular sclerotic process has gone on chiefly in the surface of the cord the so-called annular sclerosis is found, a ring of sclerotic tissue surrounding the fairly

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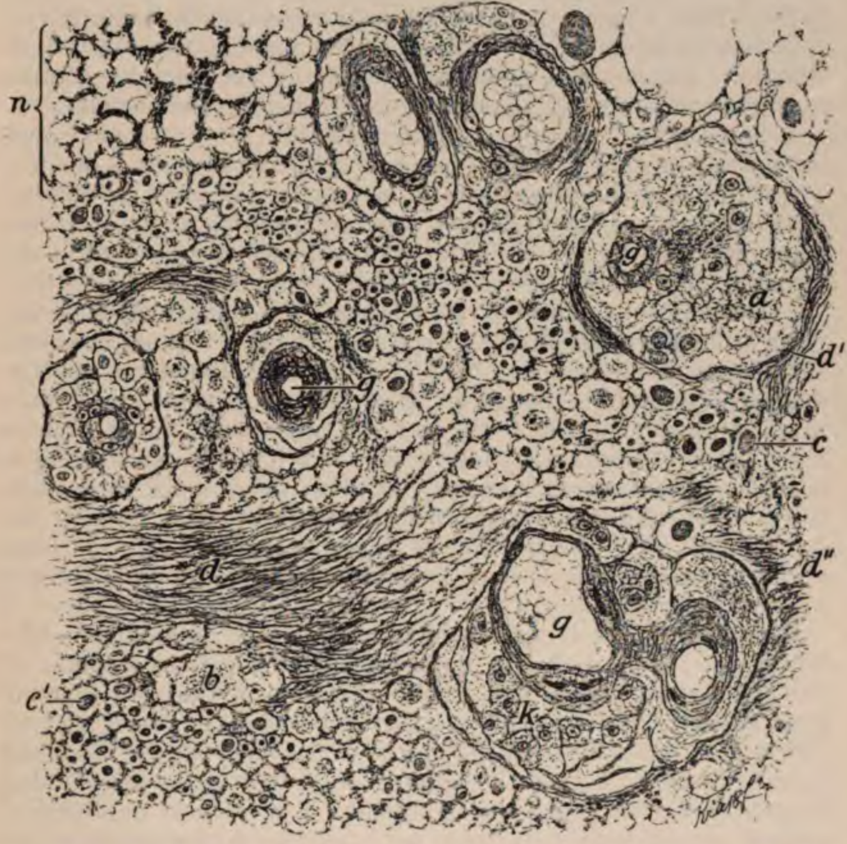
[1] F. Billings. The Shattuck Lecture, 1902.

FIG. 126.



Chronic myelitis. Swelling, degeneration, and sclerosis in the cord. *a.* Degenerated tissue. *b.* Cell in state of chromatolysis. *c, c'.* Swollen axis cylinders. *d.* Sclerotic meshes of glia. *g.* Vessels with thickened walls. *n.* Enlarged meshes of glia. (Schmaus-Sacki.)

normal fibres of the cord, the gray matter not being greatly affected. This condition is rare.

The second pathological change present in chronic myelitis is an advanced degeneration of the nerve fibres and of the nerve cells throughout the cord. This may be very irregular in its distribution; and its extent at the time of the autopsy is very often much greater than the extent of the initial sclerosis to which it is secondary. Secondary degeneration follows each nerve fibre to the end where it is interrupted at any point in its connection with its proper neurone body. Thus a small patch of sclerosis at one level of the lateral column may be followed by a long descending degeneration in that column. And a small patch of sclerosis in the posterior column may give rise to ascending degeneration as high as the medulla.

It is not always possible to determine from a microscopic examination whether the primary process in a chronic myelitis is due to a degeneration of the nerve fibres with secondary sclerosis, or whether it is due to a primary sclerosis with compression and secondary degeneration of the nerve fibres. Both conditions are found in the terminal stage of the disease. In some sections it is evident that the thickening of connective tissue is greater than that which occurs in an ordinary replacement hyperplasia. In other regions, however, the sclerosis is quite comparable to that ordinarily found in secondary degenerations. It is this mingling of the two processes in chronic myelitis which gives rise to the irregular appearance found. Enormous swelling of the axis cylinders, such as is found in acute myelitis, is occasionally observed in chronic myelitis; and granular corpuscles, the products of degeneration, are scattered everywhere through the spinal cord. The distribution of these lesions through the cord varies in different cases, both transverse and disseminated chronic myelitis being found.

Symptoms. The symptoms of chronic myelitis are such as might be expected from a chronic implication of the various columns of the spinal cord and of the various segments of the cord in their gray matter. The process is such a slow one, however, and so different in different cases that no typical course can be described in this disease. A general feeling of weakness in the legs, undue fatigue on exertion, going on gradually to a state of paralysis of greater or less degree, perhaps not sufficient to incapacitate the patient for many years, is the usual history. The paralysis is usually of the spastic type, with rigidity in the muscles, but it may be confined to a few muscles in the limb affected, and these muscles may show atrophy and reaction of degeneration. As a rule, there is in the early stage an increase in the tendon reflexes, with ankle clonus, and this may persist for several years and finally subside, and in the later stages the reflexes may be lost. Occasionally, if the lesion happens to affect the reflex arc, the knee-jerks are lost at the outset. The legs may be the only parts paralyzed for many years, or the paralysis may extend within the first year to the arms, or it may begin in the arms, but usually after several years some symptoms of paralysis are present in all the extremities. The paralysis rarely is as extreme as it is in the forms of muscular atrophy, and therefore rarely gives rise to permanent deformities or contractures. These, however, are occasionally seen.

Disturbances in the sensory sphere are very common in myelitis. These usually consist of irregular paræsthesiæ which may be followed by plaques of anæsthesia and analgesia. Pain is not a common symptom, excepting in so far as the general stiffness and weakness give rise to extreme sensations of fatigue or painful affections of the muscles paralyzed, but pain in the back is not uncommon when the myelitis is associated with a chronic meningitis. In these cases also some shooting pains in the limbs and in the body may develop.

Trophic disturbances are quite common in the course of chronic myelitis. Urticaria, irregular eruptions upon the skin, vasomotor paralysis, giving rise to flushes or streaks of pallor, irregular growth of the hair and of the nails, trophic disturbances of the joints and of the bones are not uncommonly observed in the course of chronic myelitis. In the last stage of the disease bed-sores are a distinct danger. But all these symptoms may vary in intensity, and many of them may be recovered from with proper care.

In the majority of cases as the result of a long-continued chronic myelitis the patient is reduced to a state of helpless paralysis. He is confined to the bed, the limbs are wasted and very often drawn up in a condition of contracture, extreme flexion of the thighs and legs with adduction of the thighs being present. In these cases the hands, too, become helpless, and the patient is reduced to the most abject state of paralysis. A complicating cystitis not infrequently develops, quite early in the course of chronic myelitis, as the irregular action of the bladder may necessitate the constant use of catheters which convey infection. Cystitis and bed-sores are the common cause of death in this disease, though, if the sclerosis invades the upper portion of the cord, affections of respiration and deglutition may be the active cause of death. Many symptoms referable to an implication of the sympathetic nervous system are usually observed in the course of chronic myelitis, crises such as occur in locomotor ataxia having been observed frequently. If the first dorsal segment of the cord happens to be affected irritation or paralysis of the sympathetic in the neck will give rise to pupillary symptoms.

Diagnosis. The diagnosis of chronic myelitis is to be made entirely by exclusion. When all other forms of spinal-cord disease can be eliminated, and yet objective spinal symptoms of irregular distribution are present, chronic myelitis may be diagnosticated. When the typical symptoms of the various spinal-cord affections are considered, it will be evident that they are none of them likely to be confounded with chronic myelitis. The most difficult diagnosis is from disseminated sclerosis. In this disease, however, it is to be noted that nystagmus and other cerebral symptoms appear early, of which the most noticeable are the scanning speech and the intention tremor. The development, therefore, of cerebral symptoms in the course of the case of supposed chronic myelitis will indicate that the original diagnosis should have been disseminated sclerosis. In the early stage of primary lateral sclerosis or of amyotrophic lateral sclerosis, where the only symptoms are spastic paraplegia and where no ostensible cause can be found, the supposition of a transverse chronic myelitis of the dorsal region may be entertained; but this diagnosis can only be substantiated when the development of sensory symptoms and disturbance in the action of the bladder and rectum make it probable that the lesion is more extensive than one affecting the pyramidal tracts of the spinal cord alone.

Prognosis. The prognosis in chronic myelitis is uniformly unfavorable, and the patients are destined to a life of invalidism.

Treatment. The treatment of chronic myelitis is one of care of the general health, the various symptoms of the disease being counteracted as they arise, so far as possible, and the patient kept in a state of comfort. All the measures already recommended in connection with the treatment of locomotor ataxia may be carried out in the course of chronic myelitis, and are not infrequently attended by temporary improvement. This is particularly true of hydrotherapeutic treatment and the careful application of massage. A prevention of any of the complicating diseases which cause death will be a duty in the latter stages.

SENILE PARAPLEGIA.

People of advanced years, especially those who are rather feeble, are occasionally afflicted by a slowly progressive weakness of the legs, attended by pain and sensations of numbness, undue fatigue upon exertion, and possibly with a slight degree of difficulty in the retention of urine. These symptoms give rise to the suspicion that they are about to develop chronic myelitis. They are of very slow progress and produce disturbance of walking which results in a slight dragging of the feet, or rather shuffling gait, difficulty in getting up stairs or stepping up into a carriage, but not attended by any true ataxia of movement. A great sense of weariness is usually experienced in these patients after any exertion, and they may be the subjects of senile tremors of the hands or of the head. Very many patients afflicted in this way are the subjects of chronic arterial disease or of chronic affections of the heart. The condition may develop occasionally quite rapidly, all the symptoms appearing within a few days, and making it difficult for the patient to move about at all. In my own experience the affection is more common in women than in men. It is probably due to some disturbance of circulation in the lower part of the spinal cord, either of the nature of an arterial anæmia or of a venous congestion. This seems likely because of the fact that in the majority of cases absolute rest in bed, skilful massage, the use of hot applications to the spine, followed by cool douching, heart stimulants, and general tonics result in a progressive recovery.

Autopsies are wanting to establish the pathology, as no fatal cases have been recorded.

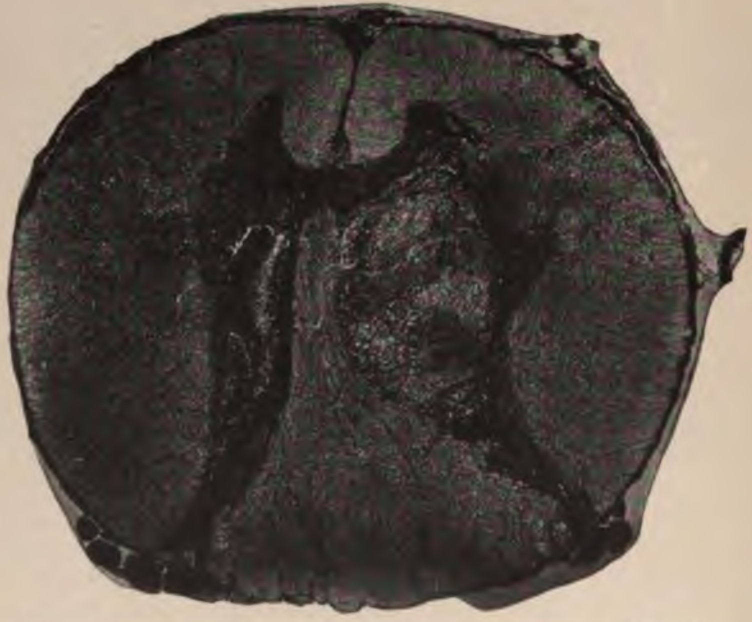
Disseminated or insular sclerosis of the spinal cord is so commonly associated with similar lesions in the brain that it will be discussed in connection with brain diseases.

CHAPTER XX.

INJURIES OF THE SPINAL CORD. HÆMATOMYELIA.

INJURIES and wounds of the spinal cord, producing a laceration of the cord and hemorrhages within it or in the spinal canal, are of frequent occurrence. The usual cause of such injuries to the cord are fractures and dislocations of the vertebræ. It is natural from the length of the organ and from its location within a narrow cylindrical bony canal that any injury to the wall surrounding it should have a direct or indirect effect upon it. And it is rare for a fracture or a dislocation of the vertebra to occur without producing immediate damage of the spinal cord.

FIG. 127.

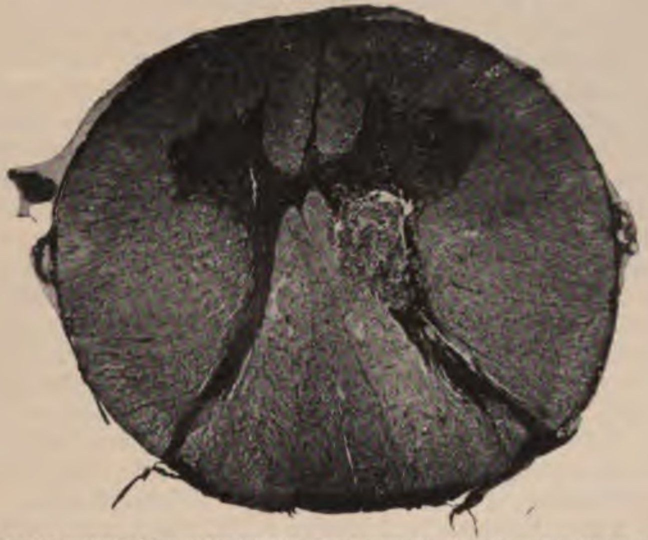


Hemorrhage in the central gray matter on the left side, due to fracture of the spine. First dorsal segment.

Pathology. Such an injury is usually attended by a compression and bruising, or by a laceration of the cord, a disintegration of its substance, and a considerable hemorrhage that perforates the cord and is followed by an inflammatory process that may intensify the disintegration or may go on to an attempt at repair by the formation of an extensive cicatrix. Fig. 127 shows the appearance of a spinal cord after a fracture of the sixth cervical vertebra. The eighth cervical segment was crushed and disintegrated, the typical form of the gray matter being obliterated and the tracts in the white matter torn across. A large hemorrhage occurred which penetrated the cord and destroyed the gray matter for some distance above and below the eighth cervical segment, being largely confined to the gray matter, especially of the posterior horn. The clot followed the line of least resistance, which is always within the gray matter. (Fig. 128.) In another case, where the fracture occurred in the dorsal region, the hemorrhage surrounded the cord above and below the destroyed segment, filling the spinal canal within the dura.

When the cord is exposed at the autopsy or in a surgical operation for the repair of the fracture, its external appearance may not be changed, and if the pia and sheath be not ruptured there may be very little evidence of the extreme destruction present. It is only upon section that the lesion is evident. Thus, in the case from which the figures are taken external inspection showed no lesion of the cord. But on cutting the cord the general disintegration and hemorrhage were visible. Bullet wounds and stab wounds of the cord cause a similar disintegration of the spinal elements and a hemorrhage without or within the cord. These are more likely to be followed by purulent inflammation, the result of septic material brought in with the bullet or knife. This inflammatory condition involves the meninges as well as the cord itself, and may go on to purulent myelitis or to abscess of the cord with total destruction in a transverse direction. Hemorrhages within the cord cause a long,

FIG. 128.



Clot in the posterior horn of the left side, three segments above the preceding figure. Sixth cervical segment.

narrow, cylindrical clot usually within the gray matter, sometimes in the posterior columns, very rarely in the lateral columns. The maximum destruction is limited to one or two segments, but the clot may perforate a number of segments, even to one-half the length of the cord. Sometimes many little clots are found at different levels. Occasionally the capsule of the cord is broken and the hemorrhage reaches the pia and infiltrates it. If the patient dies at once the clot is found surrounded by broken-down cord tissue filled with small cells and granular corpuscles—pigment and hæmatin crystals. If the patient survives a few weeks the clot may be somewhat absorbed and contracted, the tissue about it may be yellowish-red from fatty degeneration of the nerve elements, and the cells and fibres will be found in all stages of disintegration. If the patient lives for some years the clot may be entirely absorbed, leaving either a long scar of connective tissue or a long cavity lined with connective tissue—the hemato-myelo-porus of Van Gieson. Hemorrhage from the vessels of the pia mater, causing compression of the cord by a clot, may occur. Janeway has described such a condition occurring in a patient, the subject of hemophilia. I have seen one case which came on suddenly after unusual exertion.

In the patients who survive secondary degenerations develop in the cord, as in cases of transverse myelitis, above and below the injured segments; secondary degeneration also occurs in the motor nerves from the segment injured, to the muscles, and atrophy of the muscles, as in anterior poliomyelitis.

Etiology. Hemorrhage within the spinal canal or within the cord may be the result of severe blows or falls or of direct concussion. Thus I have seen multiple hemorrhages in the pia with a number of small clots outside the cord following a fall through an elevator shaft down five stories. I have also seen a hemorrhage in the conus terminalis due to the patient sitting down where there was no seat and injuring the buttocks. Hemorrhages may also occur as the result of spontaneous rupture of bloodvessels, either after great effort or after long-continued arterial disease. In one case a severe sudden bending forward of the neck without dislocation or fracture was followed by a hemorrhage within the cord which penetrated through the cervical and half the dorsal segments, causing a long tubular clot. In another case the lifting of an unusual weight was immediately followed by a hemorrhage in the lower lumbar region which destroyed the lumbar and sacral enlargement and infiltrated the cauda equina, the latter being found embedded in the clot at the autopsy.

Symptoms. When a vertebra is dislocated or fractured a displacement either of the spinous process or of the body of the vertebra can be felt, causing a double deformity. The muscles about the fractured bone are thrown into a state of rigidity, and a fixation of the back or neck in an abnormal position occurs. Any motion causes great pain both at the point of injury and in the domain of nerves which are compressed or lacerated. The exact character of the injury can only be determined by an examination with the Roentgen rays. There are some cases on record where slight fractures or partial dislocations caused no spinal symptoms. There are many cases where the spinal symptoms exceed in severity any apparent surgical injury, for dislocation may be temporary and spontaneously reduced. The surgical aspect of the case, however, is of less interest than the symptoms of spinal lesion. These depend wholly upon the position of the injury; the higher the fracture the more extensive the symptoms, for the injury usually arrests all voluntary motion and conscious sensation in the parts below the level of the lesion. Hence the level of the symptoms indicates the level of the lesion. The facts of the localization of spinal functions already presented in detail need not be reproduced here; but if carefully studied they will permit an accurate diagnosis of the position of the lesion to be reached. Particular attention should be paid to the posture involuntarily assumed by the patient in bed (see page 184), to the extent of the paralysis (see page 173), and to the distribution of the anæsthesia (see page 193).

In any case of injury of the cord a state of paraplegia is produced at once. The paralysis is complete, the limbs are relaxed and flaccid, the reflex action is much diminished, and, in cases of transverse lesion with entire division of the cord, is lost; there is incontinence of urine with retention and a paralysis of the rectum; there is often a state of priapism; there are pains in the back at the level of the fracture and in the region of the body to which the nerves near the fracture go; these pains are often attended by twitchings of the muscles which cause much discomfort; there is a condition of anæsthesia more or less complete whose upper limit is easily defined and corresponds to the level of the lesion; there is a zone of hyperæsthesia just above the anæsthetic level. The segment of the cord which is affected in any particular fracture is the one that is opposite the fractured vertebra. The researches of Reid[1] have demonstrated that there is not a uniform relation between the vertebræ and the segments of the cord. The diagram (Fig. 129) of Dejerine shows the usual relation between the various segments of the cord and the spines and bodies of the vertebræ.

Chipault has given a practical set of rules for determining the relation of the segments to the spinous processes of the vertebræ. He says: "In the cervical region add one to the number of the vertebra, and this will give the segment opposite to it. In the upper dorsal region add two; from the sixth to the eleventh dorsal vertebra add three. The lower part of the eleventh dorsal spinous process and the space below it are opposite the lower three lumbar segments. The twelfth dorsal spinous process and the space below it are opposite the sacral segments." The spinal cord ends opposite the body of the first lumbar vertebra or opposite the cartilage in the space below it. The lower half of the spinal canal is, therefore, occupied by the cauda

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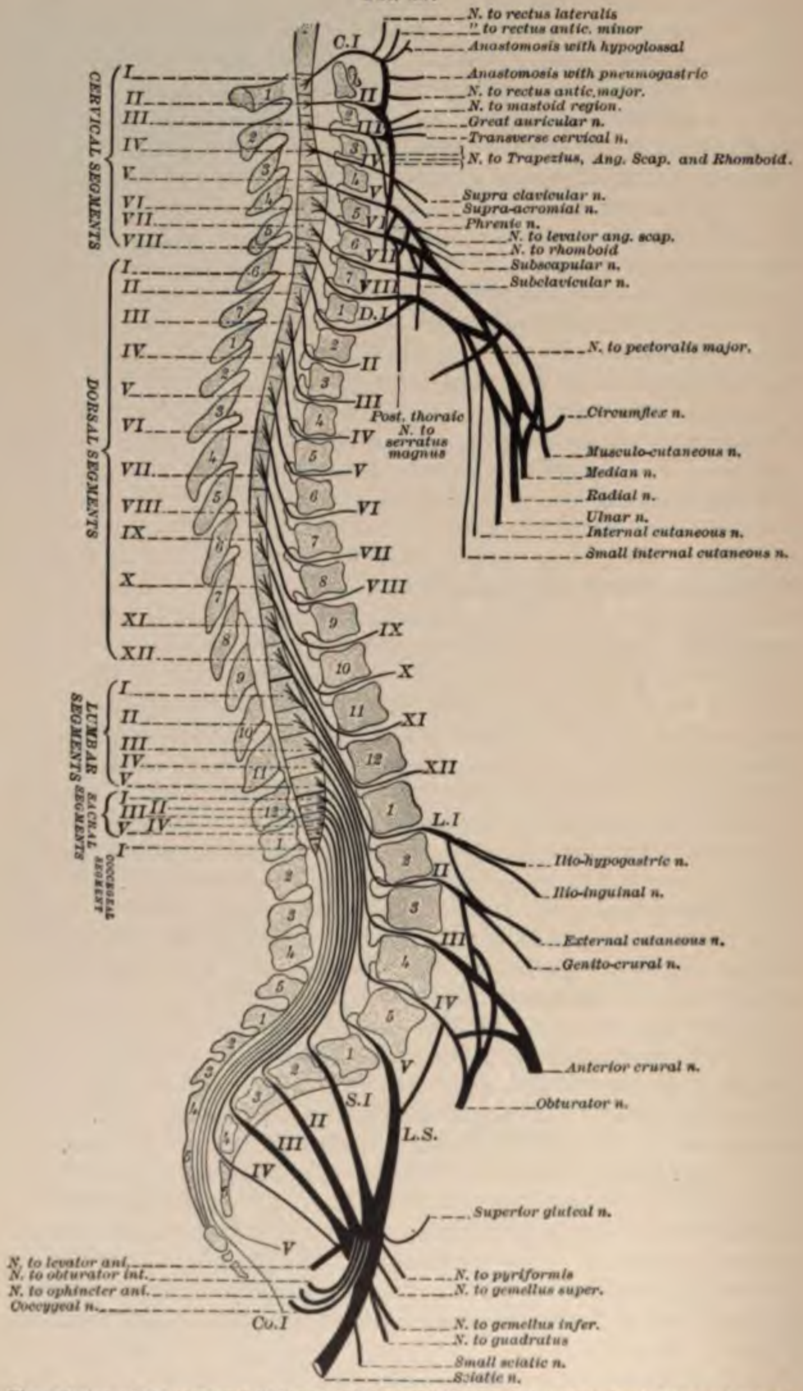
[1] Journal of Anatomy and Physiology, vol. xxiii. p. 312.

FIG. 129.



The relation of the segments of the spinal cord and their nerve roots to the bodies and spines of the vertebræ. (Dejerine et Thomas, Mal. d. l. Moelle Épinière, Paris, 1902.)

equina only. In infants the cord reaches as low down as the body of the third lumbar vertebra, but by the age of twelve years the lower limit is opposite the body of the second vertebra.

Certain special symptoms attend fractures at different levels:

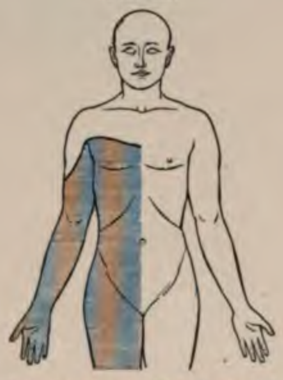
I. Fractures of the upper four cervical vertebræ are usually attended by sudden death either from involvement of the vital centres in the adjacent medulla or by a lesion of the centres of the phrenic nerves and respiratory paralysis. In a few patients who have survived for a few days pain has been felt in the great occipital nerves; the head has been held rigid, and any motion of it has been very painful; a deformity has been felt in the pharynx; a total paralysis of the entire body below the neck has occurred, and high or subnormal temperature with very rapid pulse have been present. Death always follows soon.

II. Fractures of the lower three cervical and first dorsal vertebræ are quite common, over one-quarter of spinal fractures being in this location. Divers into shallow water, laborers whose heads are bent forward upon their bodies, and persons struck by heavy objects falling on the shoulders, or who fall from a height in such a manner that the body is bent upon the head usually fracture one or more of these cervical vertebra. In such fractures either the upper or the lower half of the cervical enlargement of the cord is injured.

(*a*) When the upper part of the cervical enlargement is injured the paralysis of the arms is complete and the arms lie relaxed at the side of the patient; pain is felt in the neck and shoulders, and spasms of the arm muscles are frequent; anæsthesia is complete below the deltoid area over the shoulder-joint (Fig. 130); there is no reflex activity in the upper extremities, but there may be some reflex activity in the legs after the shock passes off, and this soon becomes exaggerated; there is usually constant priapism, and retention of urine which must be relieved by catheter; there is paralysis of the rectum; there is a marked tendency to bed-sores, respiration is wholly diaphragmatic; coughing and expiratory efforts are impossible; a high fever and rapid pulse develop; unusual sweating of the entire body occurs, and, as a rule, death follows during the first or second week. If the patient survives a state of nearly complete paralysis remains, with atrophic paralysis of the arms and spastic paralysis of the legs.

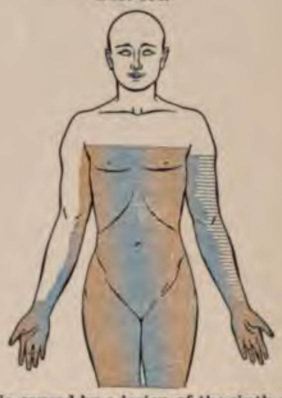
(*b*) When the lower part of the cervical enlargement is injured the paralysis of the arms is partial, the shoulder muscles and the flexors of the forearms escaping, and sometimes some of the muscles of the forearms also escape. In this form the patient lies with the arms abducted from the sides and the forearms flexed, the hands resting on the chest; the body and legs are paralyzed and flaccid, pain is felt in the hands and on the inner side of the forearms and often about the body; the anæsthesia is more marked on the inner side of the arms and forearms and is total in the hands (Fig. 131 to 133); it affects the body also, but the upper limit of anæsthesia on the body may be as low as the nipple; there is a region of hyperæsthesia on the outer side of the arm and about the chest above the nipple; there may be a temporary suspension of reflex action in the legs, followed later by exaggeration of all the reflexes; there is priapism, retention of urine, paralysis of the bladder and rectum; there is an oculo-pupillary paralysis with contraction of the pupil, failure to dilate when the neck is scratched, and a narrowing of the palpebral fissure, and retraction of the eyeball, these symptoms indicating a lesion of the first dorsal segment, but being frequently present with any injury of the lower cervical enlargement. The paralysis is not always symmetrical in the arms, and many muscles may escape if

FIG. 130.



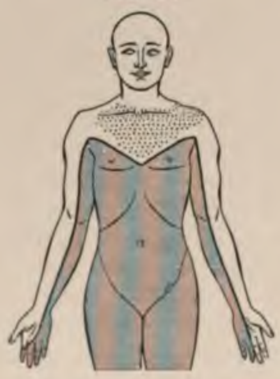
Anæsthesia caused by a lesion of the fifth cervical segment.

FIG. 131.



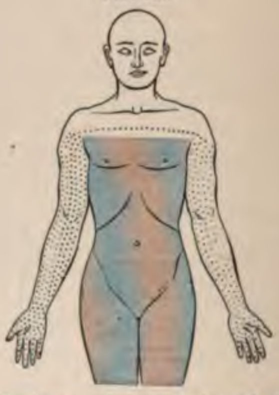
Anæsthesia caused by a lesion of the sixth cervical segment on the right side, and involving the fifth segment of the left side.

FIG. 132.



Anæsthesia caused by a lesion of the seventh cervical segment, almost symmetrical. Dotted area hyperæsthetic. (Herter.)

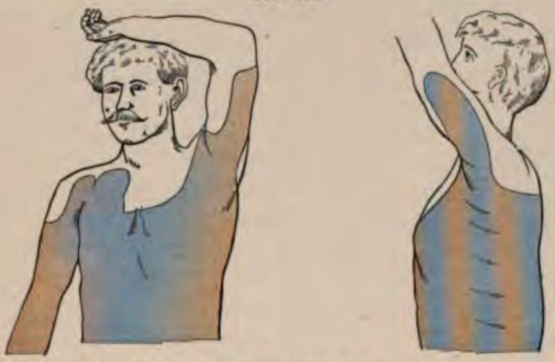
FIG. 133.



Anæsthesia caused by a lesion of the first dorsal segment. Dotted area hyperæsthetic. (Bruns.)

the lesion is low down in the enlargement. (Fig. 134.) If the patient recovers a state of atrophic paralysis in the upper extremities of greater or less extent remains and a condition of spastic paralysis in the legs.

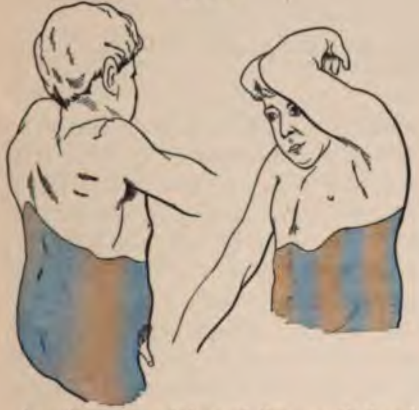
FIG. 134.



Anæsthesia of the right side due to lesion of the fifth cervical segment; of the left side due to lesion of the first dorsal segment; tongue-like extension of anæsthesia in axilla and under the arm. (Wichmann.)

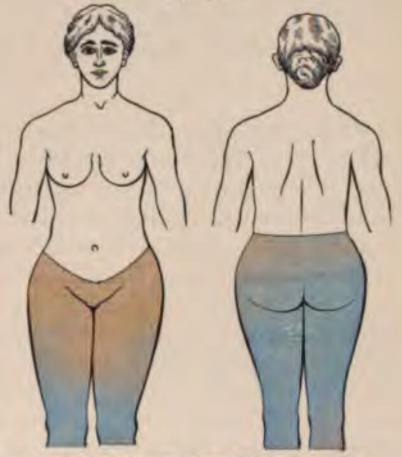
III. Fractures of the dorsal vertebræ are rather infrequent. They cause a paralysis of the legs and abdominal muscles with loss of control of bladder and rectum. There is a distinct girdle sensation often attended by pain. There is a line of anæsthesia about the trunk above which is a zone of hyperæsthesia. This line of anæsthesia is usually the most important guide to the level of the lesion, but it is not on a line with that level. (Figs. 135 and 136.) It is from three to four inches below it, for there is an overlapping or

FIG. 135.



Anæsthesia from injury of the seventh dorsal segment, showing the anæsthesia curve about the body and extending higher behind than in front. (Wichmann.)

FIG. 136.



Anæsthesia due to lesion of the eleventh dorsal segment. (Wichmann.)

anastomosis of the sensory filaments of nerves, and the intercostal nerves of one segment supply the skin directly related to two or even three adjacent segments, as Sherrington[1] has shown. (Fig. 137.) Furthermore, the sensory fibres entering the cord ascend to the two higher segments before terminating, and hence a transverse lesion of the cord does not cut off all the sensations from the nerve roots at its level. This will be understood by reference to Fig. 42. Thus in a case of fracture of the sixth dorsal vertebra the eighth dorsal segment was injured, and the line of anæsthesia corresponded to the distribution of the tenth dorsal nerve, being three inches below the level of the injury. The zone of hyperæsthesia which lies above the level of the anæsthesia is due to the fact that sensations received in segments of the cord just above the lesion which are in a state of inflammatory irritation are felt to an exaggerated degree. The girdle sensation is a consciousness of this hypersensitive skin. If the cord is not severed and is capable of transmitting some sensations through the lesion, patients often refer to this hypersensitive zone, all sensations coming up from the legs. Thus in one of my patients a pin-prick, or pinch, or touch, or a thermal sensation produced in the paralyzed and anæsthetic legs or trunk, was always felt about the level of the sixth dorsal nerve. If on the back of the body it was referred to the back; if in front, to the front, and it was always felt on the side which was irritated. This man had a transverse injury of the cord due to fracture at the fifth dorsal vertebra and had been paraplegic four years when examined. I have seen this symptom in many cases, but have not seen it described. It is of service in distinguishing total from partial lesions of the cord. In cases of dorsal injury the initial symptoms may slowly subside in part. The patient remains, however, in a state of spastic paraplegia with all the symptoms of lateral sclerosis due to the descending degeneration in the motor tracts of the cord. The danger of bed-sores and cystitis is directly propor-

FIG. 137.

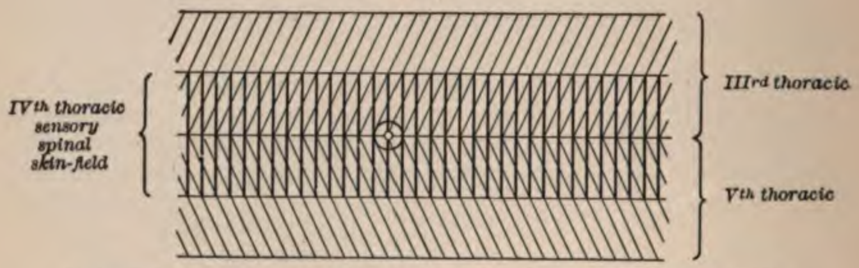


Diagram showing the nerve supply of the skin of the thorax; every part is supplied by two nerves. (Sherrington.)

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[1] Transactions of the Royal Philosophical Society, London, 1893, vol. clxxxiv.

tionate to the degree of anæsthesia remaining. Sometimes these patients live for many years.

It is in cases of dorsal injury that the syndrome due to a lesion of one lateral half of the cord, known as Brown-Séquard paralysis, is most commonly seen, although it may result from a lesion at any level. This combination of symptoms is as follows:

1. On the side of the lesion there is paralysis with rigidity; an increase of tendon reflexes; vasomotor paresis causing a temporary elevation of temperature; a diminution in muscular sense and of the sense of position of the leg; a general hypersensitive state to touch, temperature, and pain up to the level of the lesion, which may subside after a time; there is a narrow zone of anæsthesia about the body to touch, temperature, and pain, the latter two being lost a little lower than touch.

2. On the side opposite the lesion there is no paralysis; there is a slight increase of tendon reflexes; there is anæsthesia to touch, temperature, and pain up to a line passing about the body about an inch lower than the zone of anæsthesia on the side of the lesion. The accompanying diagram (Fig. 138), illustrating the course of motor and sensory fibres through a cord, which is supposed to have a lesion involving one segment on the left side only, demonstrates the manner in which these symptoms are produced. The lesion interrupts the transmission of nervous impulses of various kinds. In a patient of my own a fracture of the seventh dorsal vertebra caused this syndrome, which was partly relieved by operation. The fractured lamina that compressed the cord was removed, the symptoms gradually subsided, but even at the end of eight years there remains some stiffness of the paralyzed leg and an appreciable difference of sensation on the two sides in the legs.

FIG. 138.

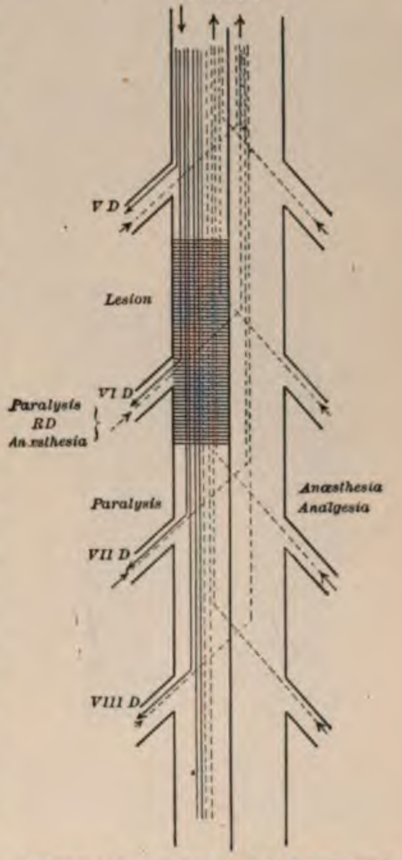
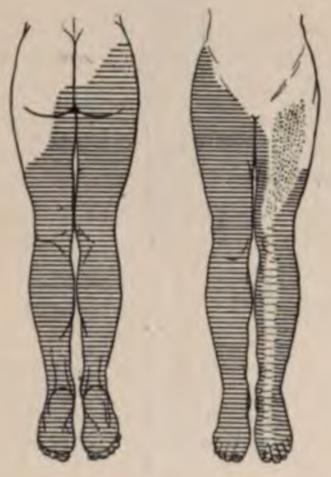


Diagram showing the effect of a unilateral lesion of the spinal cord, causing "Brown-Séquard paralysis."

IV. Fractures at the lower two dorsal and upper lumbar vertebræ are the most common fractures of the spine, over one-half of the cases being in this locality. Such fractures injure the lumbar enlargement of the cord.

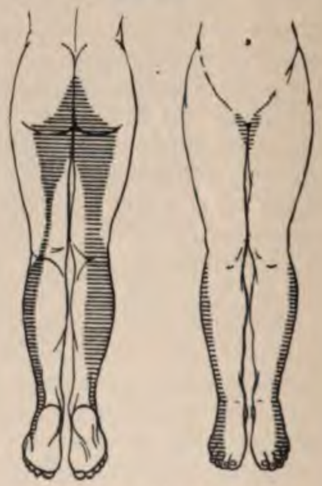
(*a*) When the upper part of the enlargement is destroyed there is total paralysis with flaccid condition in the abdominal muscles and in the muscles of the thighs, with increasing atrophy and reaction of degeneration and a loss of reflex action; there is paralysis without atrophy or change in electric reaction in the muscles of the legs and feet; the legs lie extended in bed and cannot be drawn up; there is loss of voluntary control of the bladder and rectum, with retention of urine; compression of the testicle does not cause a contraction of the abdominal muscle of the same side (Kocher's reflex); there is anæsthesia as high as Poupart's ligament about the body, and the girdle sensation is located here. Sometimes, however, when the upper two lumbar segments escape, the funnel-shaped area on the front of the thigh is hypersensitive (Fig. 139), a sense of pain is felt there, and the anæsthesia is confined to the area corresponding to the third lumbar segment and the segments below it. If these patients recover in part, as they often do, a great degree of inability remains, as the thighs are paralyzed.

FIG. 139.



Area of anæsthesia in a lesion of the lower two lumbar segments on the left side and entire lumbar enlargement on the right side. (Starr and McBurney.)

FIG. 140.

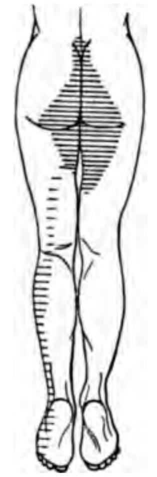


Anæsthesia after a lesion of the fifth lumbar segment. (Eulenberg.)

(*b*) When the lower part only of the lumbar enlargement is destroyed, the upper portion escaping, the paralysis is limited to the legs below the knees, or even to the peronei and feet, and these muscles are soon atrophied and show a reaction of degeneration; the thighs are drawn up in bed and the legs are flexed; the knee-jerks are sometimes present, but there is never any ankle clonus or toe reflex;

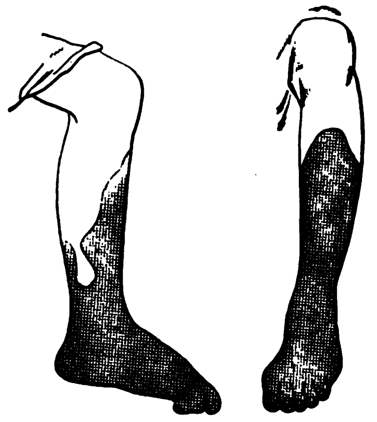
there is paralysis of the bladder and rectum, with complete relaxation of both sphincters; pain is felt in the back and in the feet or legs; the anæsthesia is confined to the back and outer part of the thigh, and is greatest on the outer part of the leg and foot, some one of the peculiar areas of anæsthesia shown in Figs. 140 and 141 being present. Sensation in the testicles is preserved. In these cases

FIG. 141.



Anæsthesia after a lesion of the first sacral segment. (Starr.)

FIG. 142.



Anæsthesia from a lesion of the fourth lumbar segment. (Wichmann.)

FIG. 143.

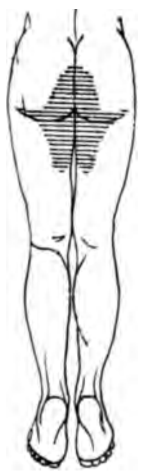


FIG. 144.

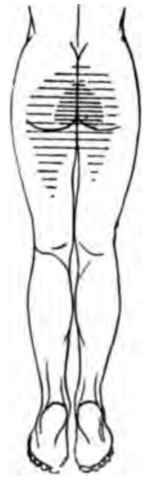
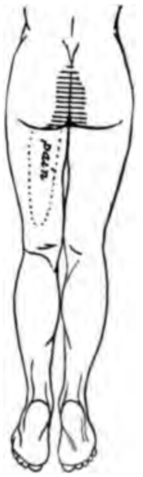


FIG. 145.



Anæsthesia after lesions of the second, third, and fourth sacral segments. (Oppenheim, Huber, and Starr.)

a recovery leaves the patient with some power of getting about on crutches, as apparatus may hold the knees and ankles firm, while the thighs are under voluntary control.

(*c*) Lesions are sometimes limited to the conus terminalis or lower three sacral segments. The most common lesion is a hemorrhage. The symptoms produced are a loss of control of the bladder and rectum, with complete relaxation of the sphincters, a total loss of sexual power, and an area of anæsthesia either limited to the anal region, including the posterior part of the scrotum or posterior half of the labia and perineum, or extending into a heart-shaped area over the sacral region. (Figs. 143 to 145.)

FIG. 146.

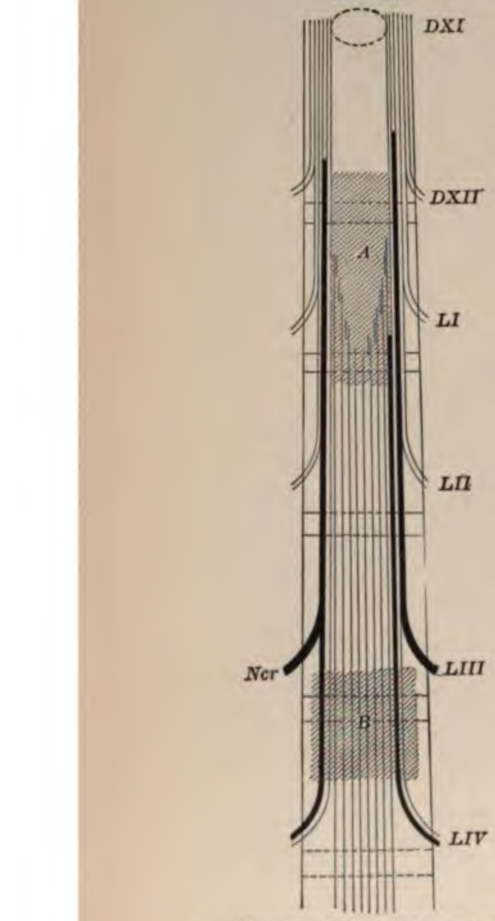
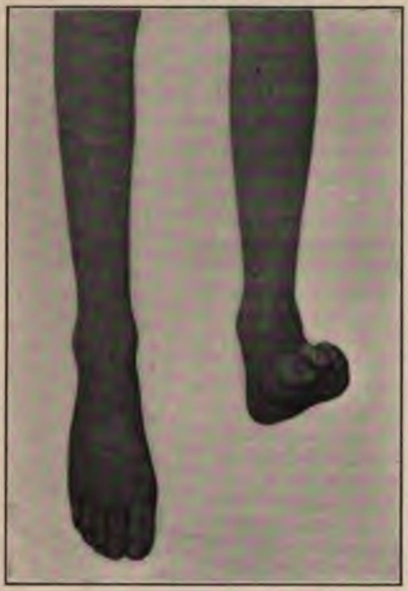


Diagram of the lumbar enlargement and the cauda equina. *A*, lesion of the lower half of the enlargement, and *B*, lesion compressing the cauda equina produce identical nervous symptoms. *Ncr.* Anterior crural nerve. (Schultze.)

V. Fractures of the lower four lumbar vertebræ or of the sacrum cause compression of the cauda equina, but no injury of the cord. The cauda equina is shown in Fig. 146. Compression of it by fracture produces very much the same kind of symptoms as injury of the spinal cord, since it is made up of the nerve roots coming off from the lumbar enlargement. When this mass of nerves is compressed it is found that the innermost ones suffer chiefly, and as these arise from the sacral segments the symptoms of injury to the cauda equina are often those of sacral lesions, viz., paralysis of the feet and peronei, loss of control of the bladder and rectum, with relaxation of the sphincters and incontinence of urine, and anæsthesia in the saddle-shaped area on the buttocks, about the anus, and on the posterior part of the genitals. The symptoms may not be equally distributed in the two legs. In one case of fracture of the fourth lumbar vertebra the symptoms were unilateral. Fig. 147 shows the drop-foot present in this patient, and Fig. 148 shows the area of anæsthesia present. No Brown-Séquard syndrome can be caused by a partial lesion of the cauda equina. The power of regeneration of the nerves is well known. Hence complete recovery is possible after cauda lesions. In the patient shown in Fig. 147, operation for the elevation of bone and removal of pressure resulted in a complete cure.

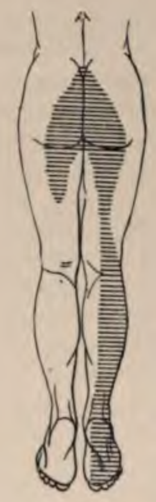
The diagnosis of a cauda equina lesion from a lesion of the lower part of the cord is not always possible from the nervous symptoms alone. The diagram of Schultze (Fig. 146) shows that two lesions may cause the same symptoms. In fractures or injuries the location of the lesion is, however, not so uncertain as it is in hemorrhage within the cord or in tumors within the spinal canal. It is to be remembered that at the site of the fracture or injury the nerves are often lacerated or compressed as they issue from the fractured vertebra. This causes intense pain or a zone of anæsthesia. The location of this pain or zone may be at a higher level than the anæsthesia produced by compression of the cauda or cord, and may often be a guide to the level of the lesion. Thus in a case of injury to the back the pain was felt on the outer side of the thigh on one side, and in this region the skin was anæsthetic, the paralysis was limited to the feet, bladder, and rectum, and there was fair sensation in the areas belonging to the fourth and fifth lumbar segments. In this case the diagnosis of a lesion of the third lumbar segment could be excluded and a diagnosis made of compression of the cauda equina and laceration of the third lumbar nerve. Symptoms of cauda equina lesions are less likely to be exactly symmetrical in their distribution than symptoms of lesion of the conus terminalis. In cauda equina lesions severe pain in the back, perineum, and genitals is more common than

FIG. 147.



Drop-foot after compression of the right half of the cauda equina, relieved by operation.

FIG. 148.



Area of anæsthesia after compression of the cauda equina. (Starr and Lloyd.)

in lesions of the cord. Bed-sores are more likely to develop in lesions of the cord.

In the description of symptoms here given the lesion has been described as a transverse one. This is the rule. But in many cases the lesion is somewhat irregular, one side or part of the cord suffering more than the other. And in other cases there occur perforating hemorrhages in the gray matter above the transverse lesion, which also give rise to symptoms. In both these conditions the symptoms are not as symmetrical as in a transverse lesion—the paralysis may be greater in one leg or arm than in the other, and the area of anæsthesia on the two sides may differ. The asymmetry of symptoms is even more marked in spontaneous hemorrhage in the cord without external injury.[1]

Another symptom which requires special mention is the extreme lancinating pain felt either in the trunk or in the extremities in the domain of the nerves which are compressed or lacerated at the point of fracture. These pains are referred to the peripheral termination of the compressed nerves; but, like the pains of locomotor ataxia, are of central origin. They often aid in the location of the lesion, as they are referred not to nerve districts but to segmental areas.

Diagnosis. The most important point to settle in any case of injury of the spinal cord by dislocation or fracture of the spine or by internal hemorrhage is the question whether the cord has been completely destroyed at any level, whether the transverse lesion is total and the cord below it is cut off entirely from the cord above. If any sensations whatever are felt by the patient in the parts below the lesion, such as pains, numbness, cramps, distention of the bladder or rectum, abdominal pain, or if changes of position given to the limbs are perceived, or if any motion in the legs is possible the lesion is not total. Even when there is total anæsthesia it is possible in some cases to cause sensations by severe irritation by needles in the paralyzed parts, and these are felt in the hyperæsthetic zone. This never occurs if the lesion is total. The tendon reflexes return soon, if lost at first, and become exaggerated in partial lesions. If the lesion is total the patellar tendon reflex is permanently lost (Miles, Bastian, Kocher), the paralysis of the legs is flaccid, and they show no rigidity on passive motion; the line of anæsthesia is more sharply defined and is absolute; skin reflexes are lost; there is greater vasomotor paralysis in the parts below the lesion; the limbs are hot, the veins distended, priapism is present or easily produced by irritation of the genitals, groins or thighs, the urine is retained, and the rectum paralyzed.

Prognosis. The prognosis in cases of injury of the spinal cord depends very largely upon the degree and severity of the symptoms presented. The higher the lesion the worse the prognosis both as to life and as to the degree of recovery.

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[1] For a full history of the cases here cited the reader is referred to the American Journal of the Medical Sciences, July, 1892, and Brain, 1894.

Inasmuch as a repair of the spinal cord does not occur, and although scar tissue may form at the seat of the lesion, no restoration of continuity of the nerve fibres is possible, and no regeneration of nerve tissue within the cord has ever been known to occur; the prognosis in spinal injuries is a very bad one. This fact should be carefully considered before any attempt at surgical interference after such injuries is undertaken. When the examination with Roentgen rays demonstrates the existence of pressure it is a matter of comparative ease to the surgeon to remove it. Such operations, however, are uniformly disappointing, for, although the surgical end desired may be attained, it is rarely followed by any improvement in the condition of the patient. Hence the prognosis in these affections is extremely unfavorable under all circumstances, and the local diagnosis of the seat of the injury is therefore a matter more of medical interest than of practical importance to the patient. In minor injuries where the spinal cord is only slightly disintegrated, and where but one or two of the main motor or sensory tracts are involved in the injury, some restoration of function may spontaneously ensue. Hence the importance of guarding against such complicating conditions as bedsores and cystitis which are the immediate cause of death in so many cases. Rest in time may result in the gradual improvement of the symptoms, and the patients may even, after medium or slight injury, be able to get about in a state of spastic paraplegia of a more or less complete degree. Gurlts has affirmed that when paralysis of the bladder and rectum continues after eight weeks a spontaneous recovery never occurs.

Treatment. The treatment of injuries of the spinal cord consists, first, of the treatment of the surgical condition which is the cause of the injury, by giving proper support to the back, by reduction of the dislocation, by correction of the fracture, if possible, and by removal of bone which is causing pressure. The question of surgical interference is often difficult to decide. No operation is justifiable during a condition of shock such as usually follows a dislocation or fracture, or when high or subnormal temperature, rapid pulse, or labored respiration are present. When, after a few days, the shock has passed off, the general symptoms may assist the decision. If there is evidence of a total transverse division of the cord an operation is useless. If the cord is only partially injured an operation may do good when it is evident that the symptoms are kept up by a permanent compression. Time may show that the compression is partly due to hemorrhage, and as this is absorbed some functions may return. The majority of surgeons advise postponing an operation until after the second week, in order to distinguish temporary symptoms due to concussion and to shock or to extradural hemorrhage from the permanent symptoms of cord lesion. To put it off much longer, however, is to wait needlessly. If there is evidence, by X-ray examination, of an existing compression of the cord by displaced bone, this should always be removed. If, however, there is no such evidence an operation should be refused. The statistics gathered by Chipault and Hahn support this view. In one hundred and sixty-seven cases Chipault found that twelve were cured, twenty-four were improved and sixty-five died. In sixty-four cases Hahn found that nineteen were cured or improved, twelve were slightly helped, and twenty-five died. Kocher agrees with these surgeons in advising strongly against any operation unless there is distinct evidence that pressure can be removed. To lay bare the cord without the prospect of relieving pressure is to expose the patient to the risk of a surgical operation which can have no result, for the nervous symptoms are due to actual permanent destruction of spinal-cord tissues incapable of repair. Hence, in the majority of cases, it is necessary to refuse operation, to keep the back perfectly quiet, and by mechanical devices which prevent motion of the vertebræ, such as cushions, plaster-of-Paris jacket, braces, extension apparatus, etc., to assist nature in uniting the broken bones in as perfect a position as is possible.

Secondly, the treatment of the nervous symptoms is by complete and absolute rest in bed for several weeks or months after the injury; by the prevention, if possible, of bed-sores, by keeping the skin in perfect order and changing the position of the patient or changing the points of greatest pressure upon the body frequently; by clean catheterization to prevent cystitis, and careful regulation of the action of the rectum by enemata. If spasmodic contractions and cramps in the muscles of the limbs at some distance below the level of the lesion is an annoying symptom, it may sometimes be controlled by the use of bromide or by the use of bromide and chloral, or by phenacetin. Pain at the level of the injury, especially in nerves that are crushed, will require the free use of morphine, which may have to be continued for a long period. The paralysis must be treated by massage and electricity if it is of the flaccid type, but where the paralysis is of the spastic type electricity will only irritate and excite the muscles to spasmodic cramps. Warm applications, packs, and massage will then be of much more service. The general health of the patient is to be regarded as well as the local condition, and is more amenable to medical treatment by drugs than is the spinal lesion.

CHAPTER XXI.

COMPRESSION OF THE SPINAL CORD.

THE spinal cord may be compressed by:

I. Caries of the vertebræ.

II. Carcinomatous growths of the vertebræ or meninges.

III. Syphilitic exudations into the spinal canal.

IV. Tumors within the spinal canal, either extradural or intradural; extraspinal or intraspinal.

V. Aneurisms of the aorta causing absorption of the vertebral bodies.

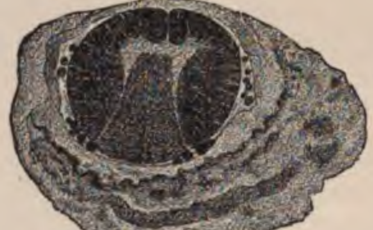
VI. Pachymeningitis cervicalis seu lumbalis hypertrophica.

Any one of these causes, acting as it does in a gradually progressing manner, and usually localized at one level, produces a slowly increasing compression of the dura, of the pia, and of the bloodvessels of the cord, and of the spinal cord and of its nerve roots, both outside and at their point of entrance into the cord.

I. CARIES OF THE SPINE.

Pathology. The most common cause of compression is caries of the spine. One of the favorite sites of tuberculous disease is the body of the vertebra, and caries starting here may extend into the laminæ as well. This caries may be and usually is attended by the formation of pus, which collects as an abscess outside of the spine and perforates, or which may press on the dura and spinal cord within the canal. The result of a tuberculous degeneration of a vertebra is a gradual softening of its bony mass, which thus removes the support of the body and allows a sinking of all the vertebra above it. As the diseased vertebra gives way a displacement forward and downward occurs. The spinous process is thus projected backward, producing the characteristic deformity of Pott's disease, viz., kyphosis. Less commonly a lateral deviation occurs. Usually in connection with such disease of the vertebra tuberculous deposits are formed between the bone and the dura mater, producing a pachymeningitis. These masses are of a caseous nature with much connective-tissue formation, but occasionally they break down into purulent débris. In very many cases of tuberculous disease of the spinal column the dura appears to offer a protection to the cord from any direct infection. However great the formation of caseous material or of pus outside of the dura the inner surface of the dura remains smooth and clean. Occasionally, however, this is not the case; the dura becomes eroded or its inner surface may be studded with tubercles, or a caseous mass may form, involving both the arachnoid and pia. Thus the membranes may be fused into a dense mass which impinges directly upon the spinal cord. Pressure upon the cord primarily affects its bloodvessels and produces a condition of ischæmia which may be sufficiently intense to go on to necrosis. Where the pressure is not very severe, this ischæmic condition, while sufficient to cause a lack of general nutrition of the cord at the point of pressure, is not enough to set up true destructive or degenerative processes; and while symptoms, such as increase of the reflexes below the level of the lesion, some degree of motor weakness and stiffness of the limbs, slight paræsthesiæ, and possibly severe pain of a girdle nature in the domain of the spinal nerves whose roots are compressed, may remain for some time, yet, if the pressure is relieved by healing of the process or by removal of the mass, they may subside and a restoration of function in the cord may ensue. In some cases, however, the pressure produces a true constriction of the cord which is visible at the autopsy.

FIG. 149.



Epidural tuberculosis. The cord is tightly surrounded by the thickened dura. Magnified with a hand-glass. (Schmaus-Sacki.)

Microscopic examination of the cord opposite the point of such pressure does not, as a rule, show necrosis rather than inflammation. There is a swelling of the axis cylinders, a segmentation of the myelin sheath with fatty drops, a disappearance of the nerve fibres, with secondary hyperplasia of the glia, and a production of sclerotic patches. The cells of the cord at this point are often atrophied and found in various stages of degeneration, chromatolysis, and vacuolization. In more advanced stages of the disease this results in a disappearance of the nerve fibres and cells, whose place is taken by small cells, small nuclei, and thickened gliomatous tissue, with an increase of connective tissue about the bloodvessels. Corpora amylacea and Deiters' cells are often found in the mass. In the terminal stage of the process the spinal cord at the point of compression is reduced to a mass of connective tissue, the nervous elements being completely degenerated. Occasionally tubercles are found along the bloodvessels extending into the spinal cord. It is evident, therefore, that the degree of the pressure will determine the degree of the degeneration. (Fig. 126, page 361.)

There is considerable discussion among pathologists as to whether the origin of the pathological process is a pressure upon the blood-vessels, producing ischæmia and necrosis, or upon the lymph channels, producing a stasis and serous effusion with œdema of the cord and secondary degeneration, or whether the mechanical compression of the nervous elements results in a primary degeneration without any vascular or lymphatic stasis. The evidence offered by different authors for the existence of all three conditions is so overwhelming as to make it apparent that all three conditions may occur. We therefore assume that the pathological process in a compression myelitis varies in different individuals. But in all the terminal lesion may be considered the same, namely, a destruction of the nervous elements and the production of gliomatous tissue of a scar-like nature at the point of compression. Fig. 124 (page 344) shows the appearance when œdematous processes are present, and Fig. 126 (page 361) shows the terminal result when scar tissue only is found.

After compression of the cord of sufficient degree to cause a disintegration of the tracts passing through it, secondary degenerations develop, and as the compression of the cord increases and the tracts in their entirety are cut off from their respective neurones, this degeneration becomes well marked. The location of these secondary degenerations has already been described on page 349, with the pathology of acute myelitis. The nerve roots lying adjacent to the cord or passing out through the caseous mass are degenerated.

Symptoms. The symptoms of caries of the spine are, first, pain, in the back in the bones that are affected, and this pain is increased by any motion or by such acts as coughing or sneezing or by sudden muscular efforts. Walking rapidly or bringing the weight of the body down suddenly upon the heels usually causes pain in the affected vertebræ. Tenderness to pressure and to heat or to electricity over the diseased vertebræ is an early symptom. Stiffness of the back soon follows. It is due to an involuntary spasmodic contraction of the deep muscles of the back, reflex in character, the object of which is to prevent any movement of the diseased bones upon one another. The stiffness is also partly voluntary, the object being to prevent any additional pain upon movement. Later on, the development of deformities of the spine and a marked kyphosis confirms the suspicion of disease of the bone. Sometimes purulent collections originating in the bone find their exit outward through the skin of the back or, burrowing widely, appear in some other point of the body, as a retropharyngeal abscess, as abscess of the mediastinum, or as psoas abscess. As the bony disease increases the patient may grow shorter, the bodies of the vertebræ being gradually absorbed.

Pain is probably the most valuable and earliest of all the symptoms of caries of the spine. It is limited to the distribution of the nerves whose trunks or roots are involved in the pathological process, and it is felt in the peripheral distribution of these nerves. Thus in the common type of caries of the dorsal vertebra pain is felt in some one or two of the intercostal nerves shooting around the body and causing a sensation of a girdle, a feeling as if a band were tightly drawn around the trunk. If the caries is in the cervical region the pain is felt in the arms or hands; if in the lumbar, in the thighs. This pain, together with the increase of knee-jerks, may be the only evidence of compression of the cord until the development of a deformity in the spine. As the disease advances the symptoms of transverse myelitis one by one appear, the motor symptoms, however, being much more common than the sensory. Thus localized atrophic paralysis may develop as the result of pressure on the nerve roots or from disease of the segment. Rigidity and weakness of the legs with increase of the reflexes develops early. The control of the sphincters may be impaired. Anæsthesia very rarely develops in compression myelitis until the cord is pretty completely destroyed, though tingling and numbness and shooting pains below the level of the compression may be present for a long time before the development of a true anæsthesia. The skin reflexes are often exaggerated. Later bed-sores may develop.

The exact symptoms of compression of the cord will depend entirely upon the level of the lesion. It is not necessary to rehearse these symptoms, as they are fully described in Chapter X., upon the diagnosis of spinal lesions, and have been mentioned concisely in the chapter upon injuries of the spinal cord (pages 371–378).

Course. The course of the disease in caries of the spine is very slow. The early symptoms may develop and continue for many months without going on to a true compression myelitis. In fact, if treatment by orthopedic measures is successful paralysis may never develop, the spine may become fixed in its deformed position, and all nervous symptoms may subside. This is the rule in childhood. When symptoms of compression of the cord develop they may progress very slowly, so that the spastic paraplegia may be present for years without incapacitating the patient. Occasionally, however, they come on rapidly. I have known a patient to be completely paralyzed in the legs within three months of the beginning of pain. A sudden giving way of a vertebral body has been known to produce immediate paralysis when the patient had been free from nervous symptoms for many months. Even when spastic paraplegia has developed fully the symptoms may gradually subside, as in a patient of Oppenheim who went on to recovery after seven years. This is a rare result. As a rule, the condition of spastic paraplegia of greater or less intensity remains throughout life, death occurring from some complication. The occurrence of caries in childhood, though followed by a long life of freedom from symptoms, may predispose a person to the development late in life of a lateral sclerosis. Gowers and Oppenheim report such cases, and I also have seen one. When caries develops in middle life the probability of serious spinal symptoms is greater than in youth, and the course of the case is usually more rapid.

Prognosis. The prognosis of compression myelitis due to caries will depend upon the possibility of removal of the cause. When the carious process is not far advanced fixation of the spine with extension is not infrequently followed by a gradual recovery, and in these cases it is not uncommon for all of the nervous symptoms to disappear. Under these circumstances it must be supposed that the pressure was not sufficient to produce any degeneration in the spinal cord, but the nervous symptoms were wholly due to an anæmia or to an œdema. In other cases where the disease makes further progress the prognosis is much less favorable. One has to think not only of the nervous symptoms but of the original disease, its progress, and of its likelihood to go on to the formation of abscesses and to a destruction of the vertebræ as well as to complete paralysis. The prognosis, therefore, in these cases is much less favorable than in ordinary chronic myelitis, and the cases go on to a fatal termination within two or three years.

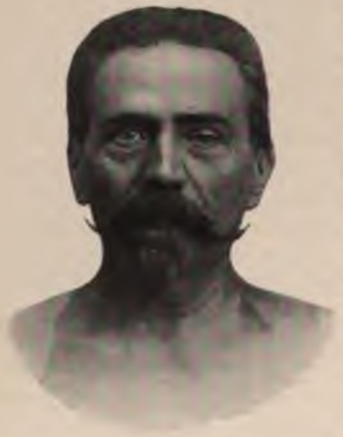
Treatment. The treatment of compression myelitis from caries consists in, first, the removal of the cause of the condition, and, second, in alleviation of the symptoms. The devices of orthopedic surgery are so numerous for the treatment of Pott's disease that they cannot be enumerated here, but the successful treatment of this condition is quite possible under proper apparatus and fixation, and in the early stage it is easy to arrest the progress of the disease, to remove the pressure from the cord, and thus to secure recovery. The treatment of the cause will often remove many of the nervous symptoms, notably the neuritic pain, the spasmodic rigidity and spasms of the legs, the cramps of the various muscles, and the paræsthesia. Pain, however, may require treatment by morphine. Spasms of the legs may be alleviated by bromide or chloral. I prefer to give ten grains of bromide of sodium every two hours than larger doses at longer intervals. If the apparatus worn will permit it, hot and cold douches and baths and packs to the spine, by increasing the circulation and nutrition in the spinal cord, are sure to be followed by a certain amount of benefit. The care of the patient's general condition must never be neglected. Fresh air, a healthy climate, the most nutritious food, supplemented by cod-liver oil and all kinds of general tonics, are fully as important as the local treatment, and should always be prescribed in addition to symptomatic remedies.

II. CARCINOMA AND SARCOMA OF THE VERTEBRÆ.

Carcinoma of the vertebræ is usually secondary to carcinoma in other parts of the body, especially of the breast. It develops very gradually in persons beyond middle life, women being more frequently affected than men. Sarcoma and osteosarcoma of the spine may develop at any age. While these tumors begin in the body of the vertebra, they extend rapidly, involving the laminæ, the ligaments, and adjacent vertebræ, and soon invade the spinal canal. Here they cause either a direct pressure upon the dura and spinal cord, or more commonly they erode the dura, and both the membranes and cord are directly infiltrated by the new-growth.

Symptoms. The symptoms are first those referable to the bony disease, pain in the spine, tenderness on pressure, stiffness and rigidity of the neck or back, and deformity. The kyphosis is usually more diffuse than in caries, as several vertebræ are affected, and the development of the symptoms is more rapid. Pain is an early symptom in this disease and is most severe and constant. It occurs in paroxysms of intense burning, shooting character, its situation depending upon the level of the tumor. It is increased by motion, by jarring, or by pressure on the spine, so that these patients instinctively avoid any movement. The nerves at their exit through the vertebra or the nerve roots within the spinal canal are directly involved in the tumor, and hence the pain persists in spite of treatment. The pain may be at first unilateral, but soon becomes symmetrical, and is attended by hyperæsthesia in the painful parts and by muscular twitchings and spasms. Herpes zoster often develops early. Symptoms of compression of the cord follow the pain. A rapidly advancing paraplegia is the rule, and as it is so uniformly attended by pain it has been termed "paraplegia dolorosa." At the level of the lesion an atrophic paralysis is produced and a spastic paraplegia in the parts below it. The level of the lesion is indicated by the extent of the anæsthesia. If it is in the lower cervical region there may be a paralysis of the cervical sympathetic nerve, as shown in Fig. 150. A loss of control of bladder and rectum develops soon, and the tendency to bedsores is very marked. The intense pain may cause a neurotic or hysterical condition, but this should never mislead, as objective symptoms are always present. The course of the case is usually rapid, and, as these cases are not open to surgical treatment, a fatal termination always follows. The duration is from three to six months. Very rarely a longer course has been observed. In one case of cervical carcinoma the symptoms of compression of the cord, with great pain in the arms, were present for three months without any local signs of the disease. It was supposed to be pachymeningitis cervicalis. This case was secondary to cancer of the liver.

FIG. 150.



Paralysis of the left cervical sympathetic nerve from carcinoma of the spine. Ptosis, retraction of the eyeball, and flattening of the face are seen.

In another case the carcinomatous character of the lesion was not discovered until after death, though the signs of compression of the cervical cord and intense pain in the occipital nerves and brachial plexus had indicated the probable nature of the disease, which was secondary to cancer of the breast. This patient for a month never made any movement without holding her head firmly with her hands. In a third case the symptoms were those of subacute myelitis of the lumbar region, with great pain in both sciatic nerves, but there was no local tenderness of the spine, or deformity. Here the autopsy showed multiple carcinomata in various abdominal viscera and a small deposit within the spinal canal involving the cord and membranes, but not eroding the bone.

In a case of osteosarcoma of the lower lumbar region there was no external evidence of the disease of the bone, and the diagnosis of a tumor of the cauda equina led to an operation being undertaken. The softened bone was found, also an erosion of the dura, and a mass of sarcomatous tissue surrounding and compressing the cauda equina.

In a case of carcinoma of the stomach a secondary cancer of the mid-dorsal region developed and the deformity of the spine became evident two months before paraplegia occurred. In this case the pain was referred chiefly to the front and sides of the body until the paralysis became complete.

It is thus evident that the local signs of the disease may not precede the nervous symptoms.

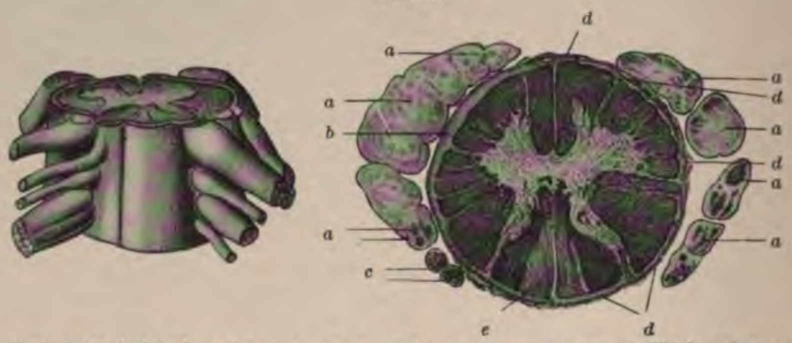
Treatment. The only treatment for this condition is palliative, and the use of hypodermic injections of morphine to give the patient relief from the pain is imperative.

III. SYPHILITIC EXUDATION ABOUT THE VERTEBRÆ AND CORD.

While gummy exostoses are not uncommon on the long bones, a syphilitic disease of the vertebræ is a rare occurrence. It does occur, however, and requires mention. I have seen it as a manifestation of inherited syphilis both in an infant and in a young man. In both cases a diffuse thickening of the tissues about the spine, in the one case in the dorsal, in the other in the cervical region, occurred, and symptoms of slowly advancing compression of the spinal cord followed. In the latter patient these advanced to well-developed paraplegia, with pains and weakness in the hands and a mild degree of spastic paraplegia in the legs. In both cases inunctions of mercury and large doses of iodide of potassium produced a gradual subsidence of all the symptoms, though in the latter case a permanent increase of knee-jerks and a slight trace of spastic gait remains.

A condition has been described of a gummy exudation encasing the spinal cord (Fig. 151), the exudation being within the dura and causing compression of the nerve roots and of the cord itself. This was not limited to one level, and therefore the symptoms produced were those of chronic myelitis. It was due to inherited syphilis. Syphilitic exudations have been observed in the tertiary stage as a result of acquired syphilis. The symptoms produced were localized swelling and signs of tumor of the vertebræ without much pain, and symptoms of compression of the spinal cord. The course was a slow one, and recovery followed treatment.

FIG. 151.



Gummy exudation about the cord and nerve roots. *a.* Gummy masses. *b.* Thickened capsule of the cord. *c.* Nerve roots. *d.* Pia. *e.* Ascending degeneration in columns of Goll. (Buttersach, Arch. f. Psych., xvii., 603.)

IV. TUMORS AFFECTING THE SPINAL CORD.

Tumors of the vertebra that press upon the cord are twice as common as tumors of the membranes and of the cord combined. Statistics show that tumors affecting the cord are relatively rare. Schlesinger found only 147 in 35,000 autopsies and 151 in 6540 tumors. The ratio to tumors of the brain is one to thirteen. Yet an added importance to tumors of the cord has been recently given by their successful removal, and hence every fact which aids an early diagnosis is of interest.

TABLE OF SPINAL CORD TUMORS.

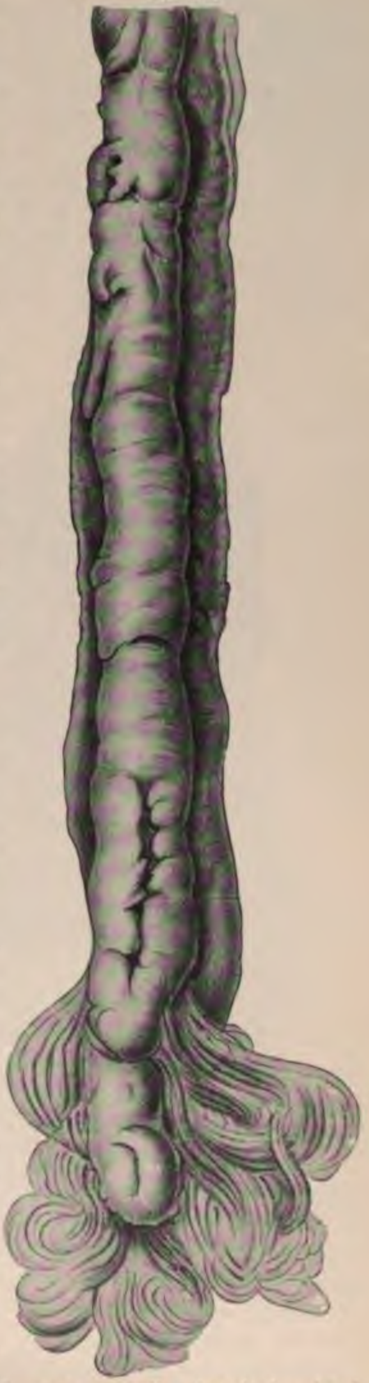
	Intradural.			Extradural.				
Variety.	Medullary.	Meningeal.	Both.	Meningeal.	Not meningeal.	Single.	Multiple.	Total.
Sarcoma,	14	53	9	17	11	80	27	107
Tubercle,	62	...	...	2	...	55	9	64
Echinococcus,	...	5	...	39	...	8	36	44
Fibroma,	...	20	2	5	...	15	18	33
Gumma,	7	4	15	2	...	19	9	28
Glioma,	20	...	...	...	...	20	...	20
Psammoma,	...	18	...	...	...	18	...	18
Myxoma,	...	7	...	4	...	11	...	11
Lipoma,	1	8	...	...	1	8	3	11
Cysticercus,	2	5	...	...	1	4	4	8
Gliosarcoma,	...	3	4	...	...	...	7	7
Endothelioma,	...	5	...	1	...	4	2	6
Melanosarcoma,	1	...	3	...	...	1	3	4
Neuroma,	4	...	...	...	...	3	1	4
Lymphangioma,	...	1	...	1	...	1	1	2
Cysts,	...	1	...	1	...	1	1	2
Cholesteatoma,	1	...	...	...	...	1	...	1
Uncertain,	13	12	2	3	...	24	6	30
	125	142	35	75	13	273	127	400

Pathology. The preceding table of Schlesinger shows the varieties of tumor affecting the spinal cord and their relative frequency, their situation, the fact that tumors are in many cases multiple, the fact that they often involve both membranes and cord together, and the fact that meningeal tumors are more common than medullary tumors.

Sarcoma about the cord has a tendency to extend rapidly, invading the membranes and the posterior surface of the cord, though more commonly compressing than destroying the cord. It is rarely encapsulated, but often surrounds the cord and can be stripped from it. But sometimes it is closely adherent, and in other cases infiltrates the cord, following the vessels and compressing and destroying the nervous elements. But the fact that such destruction is the exception rather than the rule increases the chance of success in its removal. Sarcoma is, however, often multiple, and not infrequently sarcoma of the cerebellum or of other organs is associated with sarcoma of the cord. Sarcoma often starts from a nerve root and invades both meninges and cord. (Fig. 153.)

Tubercle is more likely to develop within the cord than in the membranes when it occurs as a solitary tumor, though a tuberculous meningitis is relatively more frequent than tumor and may develop as a complication of it. Solitary tubercles are occasionally found throughout the cord, as Raymond has shown, but the usual form of tumor is a caseous mass within the cord. In some cases it appears to erode the cord, not increasing its diameter or changing its contour, and it usually begins in the gray matter, showing that the infection reaches the cord by the vessels rather than from the meninges. In

FIG. 152.



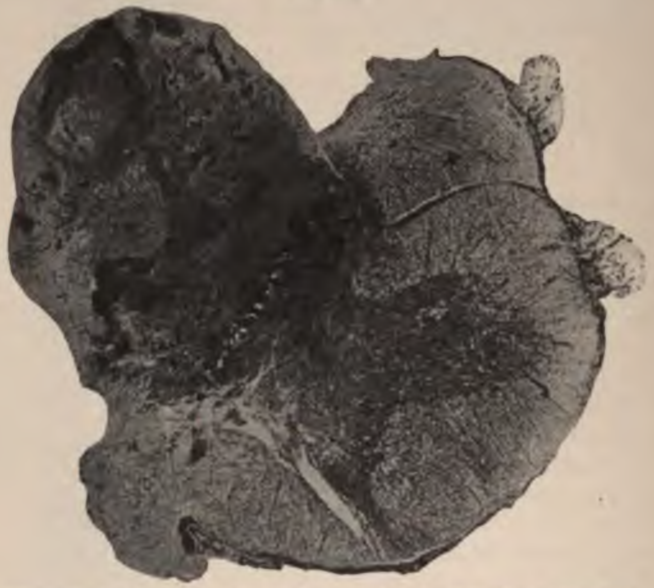
Sarcoma surrounding the spinal cord and invading the nerve roots. (Schulz.)

a few cases, as in one of my own, a tuberculous mass may lie upon the cord and compress it, and hence be easily removed.

But the tendency to recurrence is more marked in tubercle than in other forms of tumor, and the difficulty of removal, together with the danger of infection of the meninges, makes an operation much more unfavorable in this form of tumor. In my case the patient developed a general tuberculous spinal meningitis two months after the removal of the tumor, and died. Tubercles grow rapidly; hence the course of the case is one of fast progress.

Echinococcus and *cysticercus* cysts are much more common in Germany than in this country, and hence require little notice here.

FIG. 153.



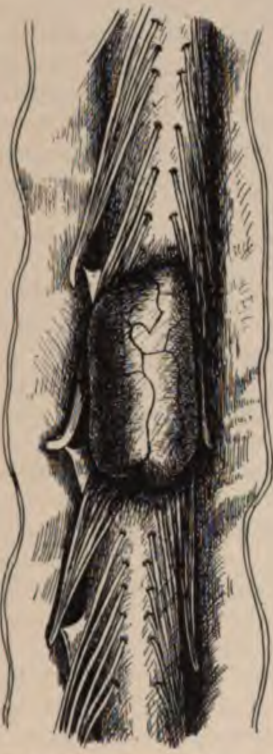
Gliosarcoma of the cord. The cord is invaded and destroyed on the left side. (Specimen furnished by E. W. Taylor and M. Prince.)

They grow rapidly, causing a progressive compression of the cord, but do not invade it as a rule. Hence the symptoms are those of compression myelitis.

Fibroma springs from the meninges, usually from the dura on its inner surface. It lies upon and compresses the cord, but does not invade it. It is encapsulated and single. It grows very slowly, and is the most favorable tumor for operation because both diagnosis and surgical treatment are comparatively simple. (Fig. 154.)

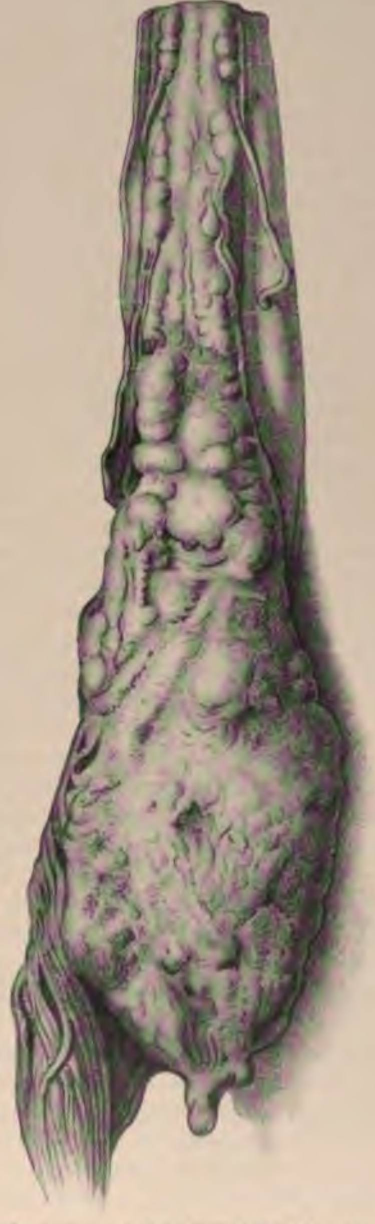
Gumma usually originates in the meninges, infiltrates them and also the cord, gluing both together in a soft mass and destroying the cord tissue completely. It is diffuse, not encapsulated, and grows very rapidly. It is often difficult to distinguish gumma from tubercle in the cord on inspection. It is a most unfavorable tumor for operation, but may yield to treatment. The treatment, however, is not followed by restoration of the spinal functions.

FIG. 154.



Fibroma lying on the spinal cord: removed by McCosh.

FIG. 155.



Fibrosarcoma of the lower half of the spinal cord and cauda equina. (Bruns.)

Glioma of the cord is not to be confounded with gliomatosis, although it occasionally complicates this disease. (See Syringomyelia, Chapter XV.) Glioma is an infiltrating tumor of the cord tissue not sharply defined or encapsulated, but causing a marked enlargement of one or two segments. It may be more extensive and involve the cord in a great part of its entire length. It does not involve the meninges. It is very vascular, but rarely contains cysts as do gliomata in the brain. It grows very rapidly and is impossible of removal. It is not infrequently associated with sarcoma, but gliosarcoma often involves the meninges as well as the cord itself.

Psammoma is a hard ovale tumor originating in the arachnoid or dura and lying upon the cord. It is sharply defined and chalky. It is not improbable that the small chalky plaques that are so common in the arachnoid on the posterior surface of the cord are the beginning of psammoma, but they rarely go on to a size that can compress the cord. A large tumor, four centimetres long and two centimetres thick, was found by Pal. This had given rise to symptoms for six years. These tumors grow slowly and, like fibroma, are easily diagnosed and removed.

Myxoma is usually intradural, sometimes extradural, but never infiltrates the spinal cord. It is single, encapsulated, vascular, and may be cystic, of slow growth, and is easily removed.

Lipoma is often multiple, is intradural, more frequently than extradural, though occasionally masses of fat are found about the back and between the vertebræ. It is a soft, encapsulated, fatty tumor of slow growth and easily removed. The other forms of tumor given in the table are so rare as to be curiosities of pathology. Endothelioma is a very small tumor, always meningeal and often multiple. Melanosarcoma occurs in the cord as a part of a general affection only. Neuroma is usually a fibroma located on a nerve root.

The diagnosis of the variety of tumor present in any case during life is rarely possible, though the facts already stated in regard to the rapidity of growth and the tendency to the appearance of tumors in other organs, or of multiple tumors of the cord may afford some hint as to the probable nature of the tumor.

Malignant tumors are ten times as frequent as benign tumors. The situation of intramedullary tumors is usually in the cervical or lumbar enlargement of the cord; that of extramedullary or meningeal tumors is chiefly in the dorsal region. If the two are taken together it appears that no part of the cord is particularly liable to tumor. Hence the situation of a tumor does not throw much light upon either diagnosis or prognosis regarding operation. Tumors are much more commonly situated on the posterior and lateral surfaces of the cord than upon the anterior surface, hence they are not difficult of access to the surgeon.

Etiology. Children are subject to tumors of the cord as well as of the brain. Of one hundred cases thirty occurred under the age of fifteen years, seventy above fifteen years. The oldest case recorded was sixty-six years of age. No age is peculiarly liable, but children are more liable to tuberculous tumors than to any other form. Glioma is next in frequency in childhood. Gumma is a disease of middle life. Other forms are equally frequent at different ages. The sexes are equally liable.

Traumatism precedes the development of tumors in so many cases as to be justly regarded as a cause. No other cause is known.

Symptoms. The clinical aspect of intramedullary and extramedullary tumors is so similar that no absolute distinction can be made in any particular case. The important thing to determine is that a tumor is affecting the cord. This is first indicated by pain of an intense kind, neuralgic, sharp, shooting, burning, and agonizing, and persistent in one locality. It is not, as a rule, increased by pressure on the nerve or attended by tender spots. There is no other disease which causes such definitely localized recurring and persistent pain as tumor of the spinal cord, and in any case in which pain is continued for over a month in one particular part of the body, there being no local disease to explain it and no evidence of spinal caries or carcinoma, a tumor may be suspected. The situation of the pain will depend on the position of the tumor, it being felt in the part of the body (not in the spine) related to the segment or nerve root first affected. Thus in a tumor of the lower cervical region the pain was felt for weeks in the hand and forearm; in a tumor of the upper cervical region, in the shoulder and neck; in a tumor of the sixth dorsal segment, in the nipple and chest; in a tumor of the tenth dorsal segment, in the abdomen and groin; in a tumor of the cauda equina, in the sciatic nerve. The pain becomes bilateral after it has persisted for a time; but this may not occur until spinal symptoms develop. Such extension to the other side is, however, a very important diagnostic symptom. There is rarely any rigidity of the spine, and bending the back does not increase the pain. The painful area is often hyperæsthetic. I have known two cases of spinal tumor which for some months were erroneously diagnosed as intercostal neuralgia, and one case was supposed to be angina pectoris.

The direct spinal symptoms are those of compression and destruction of the segments on which or in which the tumor lies. They are atrophy and paralysis of the muscles, with reaction of degeneration, loss of reflex activity in their tendons, anæsthesia in the skin, which may be attended by analgesia and thermic anæsthesia, although these affections of sensation may occur independently and without loss of touch; and herpes zoster and trophic changes in the domain of the affected nerve. The existence of dissociated anæsthesia is not always due to intraspinal tumors, as it has been found in cases of extradural tumor, but in such cases it is usually temporary.

The indirect spinal symptoms are those of spastic paraplegia of the legs, due to pressure upon the long tracts traversing the segment affected. They are rigidity and weakness with exaggerated reflexes in the legs, an imperfect control of bladder and rectum, anæsthesia up to the level of the lesion, and a tendency to the development of bed-sores. They do not occur in tumors of the lumbar region, as tumors there cause atrophic flaccid paralysis of the lower extremities.

Tumors of the cauda equina cause great pain in the sacral region as well as in all the nerves of the legs; hence the pain is more widespread in these tumors than in tumors of the cord. The paralysis which follows is an atrophic one and is attended by reaction of degeneration. It may be limited to some one of the lumbar or sacral nerves and is rarely bilateral and symmetrical. Paralysis of the bladder and rectum are early symptoms. The facts presented in the chapters upon the diagnosis of local lesions and myelitis and injuries of the cord may be referred to, as they explain more fully these various symptoms.

It is to be remembered, however, that in no other form of disease of the cord do the symptoms of transverse myelitis of gradual onset coincide with the symptom of persistent pain. When the symptoms are fully developed it is possible in some cases to elicit tenderness on pressure upon the spine over the tumor, and the patient is often extremely sensitive also to heat or to electricity in the region of the tumor. Occasionally the symptoms of Brown-Séquard paralysis appear, as in a case of gumma under my care which was gradually absorbed by treatment.

Course. The course is progressive. The duration varies somewhat according to the nature of the tumor. It is on an average about sixteen months from onset of symptoms to death. Death occurs from complicating bed-sores or cystitis.

Diagnosis. The diagnosis of a spinal tumor presents no difficulty, and should be made very early; in fact, as soon as spinal symptoms are associated with intense persistent pain in one locality. The first question which arises is whether the tumor is single or multiple. This can be determined by the location of the severe pain, for it will appear in several regions if there are several tumors, and by the development of mixed types of paralysis; atrophic rather than spastic paralysis being the type produced by each tumor in the muscles related to the segment involved.

The second question is whether the tumor is intraspinal or extraspinal. This is to be considered in part in connection with the third question, viz., what is the nature of the tumor? For if the condition of the patient, his previous history, and his present state point to tuberculosis the chance is in favor of the tumor being intraspinal. If it points to gumma the chance is in favor of its being either; if it points to carcinoma or sarcoma from an affection of other organs it may be either; if it is very rapid in its progress it may be glioma and intraspinal, or sarcoma, and either intraspinal or extraspinal. If the growth is very slow in its progress it is most likely to be a fibroma, a psammoma, or a lipoma, and in any of these cases extraspinal. All malignant tumors are more rapid in the development of all symptoms than are benign tumors and are attended by general cachexia and constitutional disturbance, which are absent in benign tumors. The fact of the previous occurrence of a tumor elsewhere and of an operation for it, which had been overlooked by the patient as having no bearing upon the case, has several times been elicited by me and thrown light upon the nature of the tumor and its secondary occurrence in the spinal cord. I have seen three sarcomata in which this history was elicited. It is evident, then, that the nature and the location of the tumor, whether extraspinal or intraspinal, is always uncertain.

The differentiation between a tumor and caries of the spine may be difficult in the early stage of the latter disease; but, as a rule, stiffness of the spine, great tenderness over the spinous process on pressure, a marked increase of pain on motion or in walking, and general constitutional evidences of tuberculous disease, swollen glands, febrile movement, etc., are present in caries and not in tumor. The appearance of kyphosis decides the question. Peptonuria is present in cases of tuberculous abscess.

The differentiation from carcinoma of the vertebra is more difficult; in fact in many cases it is impossible until the disease of the vertebra is palpable and deformity is evident, though the previous history of the patient and the rapidly advancing cachexia may point to that disease. I cannot agree with the statement of Gowers that the pain in carcinoma is greater than in spinal tumor, but in carcinoma it is more commonly felt in the spine itself as well as in the nerve distribution. Herpes zoster is rarely seen except in tumor. The examination of the blood gives little information, as leucocytosis may be present in both conditions. But examination by Roentgen rays may be of much service.

Treatment. In the early stage of pain analgesics of all kinds are to be used, but morphine will eventually have to be resorted to, as the new analgesics rarely quiet the pain sufficiently. Rest in bed cannot always be enforced, as patients may find that an upright position gives relief to the pain. Thus in one of my patients, who was found to have fibroma of the dorsal region on the posterior surface of the cord, a sitting posture leaning forward over the back of another chair was the only one in which the pain was relieved. In the majority of cases a series of inunctions of mercury combined with the administration of large doses of iodide of potassium has been tried. In cases of gumma this is curative, as I have seen in three cases, but it is to be remembered that gumma is rare (twenty-eight in four hundred cases of Schlesinger), and unless there is a previous history of syphilis no improvement should be looked for. In two of my cases precious time has been lost by such treatment.

An operation is to be attempted as early as possible after the diagnosis is reached. It should be undertaken with the understanding that it is exploratory, but it should be urged as affording the only hope. It is a bloody operation and one which takes much time and no small amount of skill; but it is not a dangerous operation if done aseptically, as shock is less frequent than in cerebral operations. The higher the tumor in the cord the greater the danger of cessation of respiration during the operation. This has occurred in two of my operations for cervical tumors, and caused much trouble, but did not prevent completion of the operation. The most important preliminary to the operation is the determination of the exact level of the tumor in the spine. It may be clear to the examiner that the tumor affects a certain segment of the spinal cord or rather that the symptoms indicate the upper level of the lesion. The level of the pain and of the anæsthesia are the most reliable indications. But it is to be remembered that the symptoms will be the same from a lesion of a segment or a lesion of its nerve roots at a point somewhat lower down within the spinal canal. Thus a tumor pressing on the roots of the sixth dorsal nerve opposite the eighth dorsal segment, where they issue from the cord, will give the same symptoms as a tumor in the sixth dorsal segment an inch higher. It is also to be remembered that the relative position of the segments and the vertebra vary, as already stated, page 369. Hence in any operation after the diagnosis of the level of the segment affected is reached and the probable relation of that segment to the spinous processes is determined it is well to make the initial incision a long one and to expose at least three vertebræ, removing their spines before exposing the cord. In one case of my own a congenital maldevelopment of one spinous process led to the count of the dorsal vertebræ being erroneous, and the cord was therefore exposed one inch lower than was intended. In the majority of patients heretofore operated upon the tumor has been found somewhat higher than was expected. It is safe to say that in the dorsal region it is four inches higher than the level of the anæsthesia on the back. When the dura is exposed over a region at least two inches long, if palpation does not reveal the tumor, it is well to lay bare another inch before opening it, and it is better to go higher than lower. The dura should pulsate, but does not do so near a tumor, especially below it. I have seen the contrast between pulsation above and no pulsation below a tumor when the dura was exposed. In all cases, even in those in which an extradural tumor is found, it is well to open the dura. In some cases a second tumor lies within. There is usually much œdema of the arachnoid about a tumor. This subsides on incision. When the tumor is found it can sometimes be easily lifted off the cord. In other cases it must be dissected away. In some cases it must be shelled out of the cord or even cut out if it is diffuse. Division of the cord should always be in a longitudinal direction, and it is better to cut wedge-shaped pieces out of the tumor than to tear it out. Intraspinal tumors usually recur. In regard to the technique of the operation, the measures of asepsis, the control of hemorrhage by compresses, the careful division of dura and pia, and ligation of bleeding spinal vessels, the proper means of stitching the dura and closing the wound, reference must be made to surgical text-books. It is an important thing to keep the patient in the prone position or on the side as long as possible after the operation to prevent undue drainage of spinal fluid. The loss of spinal fluid after an operation may be considerable without any permanent ill effect.

I have been able to collect records of fifty-eight cases of tumor of the spinal cord in which an operation has been attempted. In all of these cases the tumor was found, although in several of them it lay at a level somewhat higher than that at which it had been originally located, and in all but three of these cases it was possible to remove the tumor. Sixteen of these cases were successful, as the patients recovered not only from the operation but were very much improved some months afterward when the cases were reported. In ten of these cases operation was partially successful, the patients recovering from the operation but not being materially improved by the removal of the tumor. In all of these cases degenerations had occurred in the spinal cord. These remained after the tumor was taken away. Had the operation been performed at an earlier date in the course of the disease there is no doubt that these cases, too, would have been successful. In the remainder of the cases the operation failed to relieve, and the patients died. In several of these cases the death was due to shock, the operation being greatly prolonged and hemorrhage being excessive. With present methods of technique this appears to be avoidable. The chief danger in the operation lies in the hemorrhage from large veins in the muscles and about the bone. Such hemorrhages cannot be arrested by tying the vessels, and must be treated by pressure. If, therefore, in the course of the operation as tissues are divided, pads held in place by retractors are pressed into the wound, such hemorrhage may be to some extent avoided. Another cause of death has been meningitis of a septic nature, and this, too, may be avoided by care in the preservation of perfect asepsis.

The terminal result of an operation will depend wholly on the degree to which the cord has been compressed or destroyed. If degeneration has set in no permanent recovery can be expected; but if the operation is done in an early stage a great degree of benefit is obtained and a slight degree of spastic paraplegia only may remain. In any case a successful operation saves life and arrests the progress of the disease, even if the patient remains a paralytic. In the majority of the successful and unsuccessful operations hitherto reported the result would have been better had the operation been done sooner. A prompt surgical interference as soon as the diagnosis is made cannot be too strongly urged. In case the tumor cannot be removed a division of the posterior nerve roots is to be made above and at the level of the tumor for the relief of the pain.

Carcinoma of the spinal cord is inoperable. In sarcoma the chances are about even that the tumor can be removed. Tubercles can sometimes be taken away, even from the anterior surface of the cord, as in a patient operated on for me by McBurney; but the danger of a general tuberculous affection of the meninges subsequent to the opertion, which occurred in this case, or of a recurrence, or of development of tubercles elsewhere, is to be remembered. Fibroma, psammoma, lipoma, myxoma, osteoma, and cysts are easily removed, and do not leave any damage to the cord if taken away early.

The treatment of complicating cystitis and bed-sores is described in the chapter on myelitis.

V. ANEURISM OF THE AORTA.

An aneurism of the aorta may erode the vertebræ and produce a gradually increasing pressure upon the spinal cord. In some cases the physical signs of such an aneurism are present, and there is no difficulty in the diagnosis. In other cases, however, as in one of my patients, the aneurism lying deep in the mediastinum upon the diaphragm cannot be detected until the pressure symptoms appear. These resemble the symptoms of spinal tumor, the compression of the nerves and of the cord producing similar pain and paraplegia. There is sometimes, however, a difficulty of movement, especially of rotation of the spinal column, which does not occur in spinal tumors, and the symptoms may suggest carcinoma of the spine or caries of the spine rather than tumor, although no deformity appears until the body of the vertebra is eroded. The appearance of kyphosis in such a case is not accompanied by any thickening of the tissues about the spinous process of the vertebra or about its laminæ, and there is less tenderness of the spine than in these diseases. In any case, however, in which symptoms of compression myelitis appear the possibility of an aneurism is to be considered and its physical signs looked for.

VI. PACHYMENINGITIS CERVICALIS HYPERTROPHICA.

Pathology. This disease, first described by Charcot, presents all the symptoms of a compression myelitis of the cervical region of the spinal cord of rather rapid progress. The lesion consists of a fibrous thickening of the dura mater with consequent compression of the cord and especially of the nerve roots in their passage through the dura. As a result of this thickening of the meninges there is usually a secondary annular myelitis in the periphery of the cord at the point of implication; hence the affection is really a meningomyelitis of the cervical enlargement. In chronic fibrous pachymeningitis hemorrhages within the dura or in the newly-formed layers of tissue upon its inner surface, such as occur in pachymeningitis of the brain, are not uncommon. The thickened dura not uncommonly becomes adherent to the pia, layers of connective tissue uniting both, and attendant upon these changes there is usually thickening of the bloodvessels. The pressure of the newly-formed tissue is exerted chiefly upon the nerve roots as they pass through the dura, and these are found embedded in the fibrous mass.

If the process does not cease with the mere thickening of the dura, connective tissue is produced along the lines of the bloodvessels into the cord. The result of the compression of the spinal cord and of its vessels is a degeneration of its tissue. There is also frequently a formation of gliomatous masses and the occurrence of secondary degenerations. In the majority of the cases the disease is of syphilitic origin. Changes similar to those described in compression myelitis follow.

Symptoms. The symptoms of this disease are identical with the symptoms of chronic myelitis of the cervical region of the cord. They differ somewhat in their distribution, according as the upper half or lower half of the cervical enlargement is first involved, and they have been divided by Charcot into several stages: first, the stage of pain; second, the stage of paralysis with contractures; and third, the stage of spastic paraplegia secondary to the compression.

The pain in the affection is felt in the back of the neck, possibly in the back of the head, and is attended by great stiffness of the neck with a tendency to bending forward of the head and difficulty on any lateral motion. Pain is also felt in the distribution of the nerve roots of the lower or upper half of the cervical enlargement, namely, in the hands or in the arms. Such pains are of an intense neuralgic character, as in any condition of compression myelitis, and are attended by anæsthesia and paræsthesia and sometimes by muscular spasms or tremor. After a period of two to five months symptoms of paralysis appear in the muscles, with atrophy and reaction of degeneration. These may be limited to the ulnar and median distribution if the lower half of the cervical enlargement is invaded, or to the musculospiral distribution if the upper half is involved. Charcot describes certain deformities of the hand and wrist due to paralysis (*main en griffe*), but these are in no way characteristic of the disease, inasmuch as they may be present in any affection producing paralysis of either the extensor or flexor muscles upon the arms and forearms, with consequent contracture of their opponents. Thus drop-wrist or claw-hand may be symptoms appearing in this disease. If the lower part of the cervical enlargement is affected there may be paralysis of the sympathetic nerve. If the affection does not come to a standstill, and the pressure affects the spinal cord to a degree sufficient to cause a degeneration of the tracts passing through it, a spastic paraplegia gradually develops, constituting the third stage of the affection. Such a condition may be of long duration and slow progress, patients having been observed in whom it lasted twenty years.

I have seen several patients in whom it seemed probable from the symptoms and course that a hypertrophic pachymeningitis of the lumbar region was present. I have no pathological proof to offer, but I am convinced that it will be found. The symptoms resemble in character those described as occurring in pachymeningitis of the cervical region, but are manifested in the lower extremities, pain, rigidity, and paralysis with talipes developing slowly and being of long duration.

Prognosis. The prognosis is fairly good if the disease is diagnosticated in its early stage.

Treatment. The frequent application of the actual cautery to the back of the neck, with the use of inunctions of mercury and large doses of iodide of potash, is not infrequently followed by a cure. This will always awaken the suspicion that the lesion is syphilitic in origin, though such suspicion may not be confirmed by pathological observation. Applications of tincture of iodine to the neck and back sometimes give relief. Antineuralgic remedies will be required, and for the treatment of the paralysis such measures as are described for the treatment of lesions of the brachial plexus and of spastic paraplegia may be employed.

The impossibility of stripping off the thickened membrane from the cord without producing laceration of the cord and excessive hemorrhage renders surgical treatment impossible.

CHAPTER XXII.

LANDRY'S PARALYSIS. ACUTE PROGRESSIVE PARALYSIS.

THIS disease, first described by Landry in 1859, is an acute ascending paralysis attended in many cases by febrile symptoms and constitutional disturbances that, in the light of our present knowledge, indicate an acute infection. It begins in the form of paralysis of the lower limbs and ascends rapidly to the body, to the arms, and to the respiratory muscles, causing death within a few days, either from respiratory paralysis, from disturbance of the power of swallowing, or from heart failure. It is very rare for the disease to come to a sudden standstill and for recovery to ensue.

Ever since the original description of Landry observers all over the world have occasionally seen cases corresponding pretty closely to the description given by Landry, excepting that in some cases sensory symptoms of the nature of a rapidly advancing anæsthesia have been present associated with the paralysis. More careful observation of temperature than was customary in Landry's time has demonstrated the fact that in the large majority of cases a febrile movement accompanies the disease. Such febrile movement was present in a case under my own observation. In this case the general appearance of the patient, the enlargement of liver and spleen, a condition of jaundice, vomiting, diarrhœa, and acute nephritis all suggested strongly a condition of infection. The paralysis became complete in the course of a week, when the patient died.

Westphal described a case quite similar in its rapid course to that of Landry's original case, but in it the disease was a descending rather than an ascending one in its progress. Hence the ascending character of the affection as described by Landry must not be considered essential to the diagnosis. In a few cases the escape of the bladder and rectum has not been complete, and incontinence of urine and feces has occurred. In a few cases, where the disease has lasted beyond a week, reaction of degeneration has developed in the muscles; hence the various diagnostic signs of the disease as laid down by Landry have gradually been eliminated, and we have to speak of Landry's paralysis as included under acute progressive total paralysis.

In the majority of the cases recently recorded the distinctly infectious appearance of the patient, as already mentioned in my case, has been prominently noticed, and it seems at present to be the general consensus of opinion that in Landry's paralysis we have an acute infectious disease which produces rapidly advancing paralysis.

Treatment. The frequent application of the actual cautery to the back of the neck, with the use of inunctions of mercury and large doses of iodide of potash, is not infrequently followed by a cure. This will always awaken the suspicion that the lesion is syphilitic in origin, though such suspicion may not be confirmed by pathological observation. Applications of tincture of iodine to the neck and back sometimes give relief. Antineuralgic remedies will be required, and for the treatment of the paralysis such measures as are described for the treatment of lesions of the brachial plexus and of spastic paraplegia may be employed.

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Pathology. The lesions found in various cases of Landry's paralysis have differed widely. The early observers found no lesions, but their methods of observation were very imperfect, and hence their conclusions cannot be accepted, especially as later investigators have almost uniformly found some changes in the nervous system. Many pathologists have found changes in the spinal cord consisting practically of a widely disseminated acute myelitis, with vascular and perivascular inflammatory conditions, with acute degenerative processes, and with all the characteristic appearances seen in cases of very acute myelitis. Other equally reliable observers have considered Landry's paralysis a multiple neuritis and have described numerous changes in all of the peripheral nerves and in the nerve roots.[1] In these cases no changes in the spinal cord have been found. Unfortunately, as yet bacterial examination of this disease has failed to reveal the existence of any special germ, though in recent cases various micro-organisms have been discovered: typhoid bacilli, pneumococcus, staphylococcus, diplococcus, etc. The probable theory, however, is that it is a disease of acute infectious origin, the germ or toxin of which affects the entire nervous system both in its periphery and in its central organs. In some cases the lesion is found in the nerves; in others in the neurone bodies in the spinal cord; in others, still, in the pons and cerebral axis, the specific action being greater on the motor than on the sensory portion of the nervous system. The cortical neurones escape. This hypothesis would give an explanation for the rare cases of acute bulbar palsy or acute ophthalmoplegia followed by descending paralysis, the majority of which go on to a fatal termination in a very short space of time. The causation of the disease is more likely to be a specific infection, inasmuch as autopsies in later cases have demonstrated changes in the viscera, the liver, the spleen, the kidneys, and the lymph glands such as are commonly found in other acute infectious diseases.[2] Its causation also finds a certain amount of support in the fact that a number of cases of Landry's paralysis have developed in the course of or subsequently to other acute infectious diseases.

Symptoms. The disease begins suddenly and affects, first, the muscles of the lower extremities, producing a rapidly increasing flaccid paralysis with total disability to move the muscles voluntarily. There is no tremor, no contracture, no spasm or cramp in the muscles, and percussion of the muscles does not produce any muscular contraction. There is a loss of tendon reflexes. The electrical reactions are not changed, but this is probably because the duration of the paralysis is not sufficiently long to warrant the appearance of a reaction of degeneration. There may be numbness in the legs, but there is rarely pain. The paralysis advances rapidly from the legs to the thighs, then to the muscles of the trunk, so that the patient can no longer retract the abdomen or sit up in bed; it then advances to the muscles of the back and of the thorax and of the arms, until finally all the muscular system up to the neck is paralyzed. It then advances to the muscles of deglutition and of speech and to the face, so that an absolutely total paralysis of the entire muscular system of the body, including that of the eyes, may occur before death. When the arms are affected the paralysis begins in the fingers and creeps up the arm as it did up the leg, and here, too, there is no tendency to contracture or deformity. The limbs are wholly given over to the action of gravitation, muscular tone being abolished. The bladder and rectum usually remain in a normal condition, though in a few cases they have been paralyzed. Sensory disturbances in some cases do not appear at all. In other cases the general sense of muscular fatigue and very intense feelings of numbness and formication may be felt, but examination usually fails to reveal any marked anæsthesia. The patient's consciousness remains clear to the very end in the majority of cases. According to Landry's account of the disease, it may run its course in some cases within three days, in others it may be longer, up to two weeks, and a large majority of patients die. In a few cases the disease comes to a standstill; remissions occur, which may be followed by an exacerbation and death, or by a slow recovery.

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[1] Bailey and Ewing. New York Medical Journal, July 4, 1896.

[2] T. Diller. Journal of Nervous and Mental Disease, October, 1902.

The prognosis is very grave, and the treatment can only be of a general character, such as would be used in any acute infection or in acute myelitis. Ergot has been recommended in large doses, but this seems to be an empirical suggestion with no rational basis. Antiseptic treatment by hypodermic injections of carbolic acid or of formalin injections into the veins might be tried.

CHAPTER XXIII.

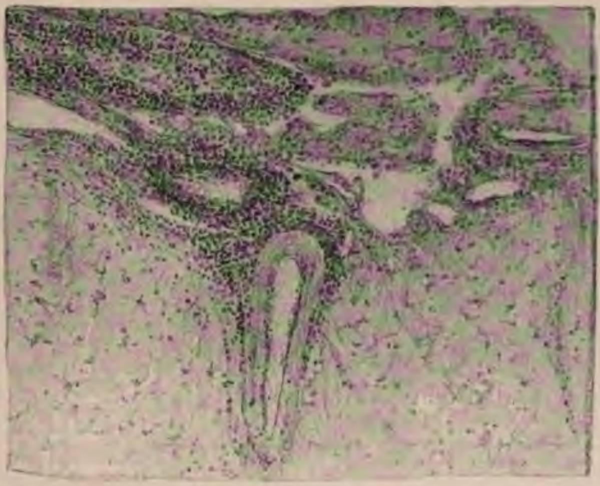
SPINAL MENINGITIS.

Etiology. Acute meningitis limited to the spinal cord develops after various forms of disease of the spinal column or of the dura mater, especially after tuberculous and malignant disease. It may occur as the result of any form of injury of the spinal column or of the spinal cord, especially if that injury involves a laceration of the surface and opens the way for the ingress of germs. Acute spinal meningitis may also develop as a complication of acute myelitis and myelomalacia from any cause. It may also arise as a complication of bed-sores, the septic agent having direct access to the meninges, either through the vessels or along the nerve trunks. It may develop also by extension of a meningitis of the base of the brain, especially after otitic meningitis. The most common cause of acute meningitis is the presence of some infectious agent in the blood as the result of pneumonia, typhoid fever, rheumatism, furunculosis, tuberculous disease anywhere, or any of the infectious diseases, or septicæmia. Exposure to cold is mentioned as a possible cause, though this is questionable.

Epidemic cerebro-spinal meningitis is described in Chapter XXXVIII.

Pathology. On opening the spinal canal an accumulation of fluid is found between the dura and pia, infiltrating the meshes of the arachnoid, and this fluid may be purulent. Here and there on the surface of the cord are collections of pus or of thick fibrin and plastic material and gelatinous masses. Fibrinous exudations upon the surface of the cord in the pia are evident at places. Thus the usual proofs of an inflammation of a serous membrane, serum, fibrin, and pus are found irregularly scattered, or filling up all the meshes of the arachnoid and covering the pia. The bloodvessels are distended with blood, and small hemorrhages may be found. There is a close adhesion of the pia to the cord, and frequently also to the dura. Meningitis may extend to the nerve roots at their points of exit through the membranes. On microscopic examination an extensive congestion of the finer bloodvessels of the meninges and of the arteries entering the periphery of the cord is evident, and there is an exudation of cells about the bloodvessels both in the pia and in the periphery of the cord. Capillary hemorrhages are frequently observed in the pia and within the cord, together with masses of fibrin and collections of pus. Œdematous swelling of the various layers of the pia and the production of connective tissue, with a marked thickening of the pial layers, are generally present. Changes are uniformly observed on the nerve roots and also in the spinal cord. The nerve roots show swelling of the axis cylinder and destruction of the myelin sheath, with all forms of degeneration in the nerves. There is also a congestion of the vessels of the perineurium and endoneurium and an exudation of leucocytes and small cells. The spinal cord is usually affected to the depth of one-half centimetre from the surface on all sides. Fine, small-cell infiltration is evident along the bloodvessels, and many leucocytes are found within the cord. The vessels are distended and occasionally ruptured. Swelling of the axis cylinders of the white columns, segmentation of the myelin, and general degenerative processes in the nerve fibres of the cord are present, and these appear to be more intense near to the bloodvessels, and sometimes can be found at a considerable depth from the surface of the cord, especially along the larger arteries. Thus wedge-shaped regions of degeneration, apex inward, may be discovered in the cord. These changes are usually more intense in the posterior and lateral columns than in the anterior columns. There is usually a tendency to an increase in the glia and to a connective-tissue production, and if the condition goes on to a chronic stage small plaques of connective tissue and sclerosis may be produced. Similar changes are observed in the medulla oblongata if the disease extends upward to the brain. If the disease does not prove fatal there may be left, as a result of an acute meningitis, a chronic permanent thickening of the pia mater with adhesions to the dura mater and with sclerosis of the spinal cord and chronic degenerative processes in the nerve roots. In the

FIG. 156.



Acute meningomyelitis. Section through the anterior border of the spinal cord and meninges, showing infiltration of the tissues by inflammatory products. (Marinesco, International Medical Congress, Paris, 1900.)

case of the anterior nerve roots these may extend outward to the peripheral nerves.

Symptoms. Acute meningitis usually develops suddenly with fever, chill, and marked constitutional disturbances, with all the phenomena of a febrile movement—malaise, nausea, and diarrhœa—and in addition, with local symptoms referable to the spinal cord and especially to irritation of its surface and nerve roots. These symptoms consist of severe pain in the back and radiating pains about the body and down the limbs, which are greatly increased by movement or by pressure or by any irritation of the surface. They are soon followed by a condition of stiffness of the back and of the neck, so that the entire body is held rigid, and all attempts at movement are resisted because of the increased pain thereby produced. An actual rigidity of the muscles of the back of the neck can be felt, and this may increase to the point of opisthotonos, especially in children. The pains of meningitis are of a neuralgic character and are very constant and frequent. They shoot from the back around to the front of the body, and they shoot up and down the arms and legs. They are attended by hyperæsthesia and hyperalgesia of the skin, so that all touch or handling of the surface is unendurable, and the patients very often are unable to bear the weight of the bed-clothing, which has to be supported by a basket arrangement in the bed. This hypersensitiveness is often attended by spasms of the muscles and twitchings of the limbs, and these cramps are often very painful. Kernig has noticed that if these patients are made to sit up in bed the legs are always drawn up, with flexion of the knee. The leg cannot be extended on account of the contracture of the flexor muscles. In the course of a few days, as the result of the inflammation and degenerative changes in the nerve roots, anæsthesia may develop in the parts that have been painful, and in some cases paralysis with atrophy develops in the muscles. This is usually noticed first in the legs, and is subsequently attended by an increase of the knee-jerks and the development of ankle clonus. Later on, if the paralysis becomes extreme, the reflexes may be lost. Within a few days of the onset disturbance in the action of the bladder develops, there being, frequently, retention of urine, requiring the use of a catheter, and obstinate constipation; later on incontinence may ensue. A tendency to the development of bed-sores is observed in all cases, and the skin must be cared for by constant change of the position and by proper support upon an air-bed or water-bed. Vasomotor disturbances are common in the shape of flushings or sudden pallors of the skin, and at any point upon the body a red line will appear after the skin is irritated by a sharp object, such as a pencil or nail (*tâches cérébrales ou spinales*).

The course of the case is often a rapid one, and within a week implication of the muscles of respiration or an affection of the pneumogastric nerve produces death from respiratory or heart failure. It may be preceded by very rapid, irregular respirations, by Cheyne-

Stokes respiration, or by intermittent, irregular pulse. In the majority of the cases the meningitis extends to the cranial cavity, and cerebral symptoms, convulsions, and cranial nerve palsies develop just before death. In other cases the progress of the case is less rapid, and two or three weeks pass before the symptoms become severe enough to give rise to alarm, and the patient may, even after the most severe symptoms, gradually recover, though frequently a state of chronic meningitis is left in which paralyses, muscular atrophies, irregular plaques of anæsthesia, and disturbance in the action of the bladder and rectum remain for many months.

Diagnosis. The diagnosis of acute meningitis is much aided by Quincke's puncture. This should be done with strict care as to asepsis, but without an anæsthetic. Local anæsthesia with chlorethyl spray may be used. It is performed by laying the patient upon the side and bending the body as far forward as possible, the thighs being flexed. A hollow needle one millimetre in diameter is then thrust between the third and fourth lumbar spines in the middle line directly into the spinal canal, its direction being a little upward in order to avoid the lamina. The fourth lumbar spine is on a line connecting the crests of the ilium. It may be thrust from three to eight centimetres before reaching the spinal canal, according to the age and fatness of the patient. When the stylet is withdrawn the cerebro-spinal fluid will flow out of the needle, either in a gush (if there is a great increase of pressure) or drop by drop (if there is little pressure). If it does not flow freely the flow can be increased by placing the patient in an upright position. From a few drops to fifty cubic centimetres may be withdrawn without any ill effects. The wound is to be closed with collodion. In cases of meningitis bacteria of various kinds have been found in the serum—tubercular bacilli in tuberculous patients; the streptococcus aureus or pyogenes in septic cases; the pneumococcus in other cases. The mere withdrawal of the fluid where the intraspinal pressure is great is followed by great relief of the symptoms, notably the pains and spasms of the back, and Quincke's puncture may be done not merely as a diagnostic but also as a therapeutic measure.

Prognosis. The prognosis in acute meningitis will depend somewhat upon the cause, and is more unfavorable in tuberculous cases than in purely septic cases. The meningitis following pneumonia and typhoid fever usually recovers.

Treatment. The treatment of meningitis consists in absolute rest in bed. An air-bed or water-bed should be employed early on account of the tendency to bed-sores. It is preferable for patients to lie upon the sides rather than upon the back. Cold-water applications, or ice-bags, should be applied to the spine. Blisters or the actual cautery may be applied along the back. Cupping also may be used, either wet or dry, according to the general condition of the individual. The general constitutional disturbances must be treated by laxatives and by antipyretics. The pains require sedatives, either bromides in large doses or bromide and chloral together, recourse being had to opium if the analgesics, phenacetin, etc., fail.

CHRONIC MENINGITIS.

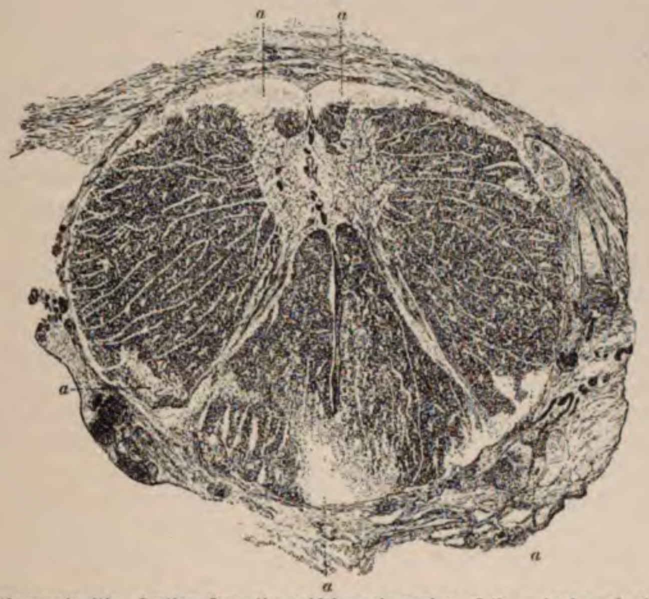
Pathology. A few cases of chronic meningitis begin in an acute process, but the majority develop as a chronic syphilitic inflammation from the start. In chronic meningitis there is no production of pus and very little fibrin, but simply a connective-tissue formation with or without an increase of serum. In all cases the result is a congestion and distention of the bloodvessels, a leucocytosis and round-cell infiltration of the membranes of the cord and of the nerve trunks, and a thickening of all the tissues involved. A chronic degenerative process is usually set up in the spinal cord, especially in the periphery of the cord, giving rise to a chronic meningomyelitis. In fact, the majority of cases of chronic meningitis are associated with chronic myelitis of low grade and slow course.

In chronic meningitis of syphilitic origin gummy exudations or gelatinous masses are found all through the meshes of the pia mater and arachnoid, and very often surround the nerve roots. Thus in the case reported by Buttersack (Fig. 151) the gelatinous mass surrounded both the cord and the nerve roots and was attended by a round-cell infiltration of the cord itself. In syphilitic meningitis the bloodvessels are thickened, both veins, arteries, and capillaries, and the lymphatics show deposits in their walls and an infiltration with small cells. The changes in the bloodvessels accompanying a syphilitic meningitis are those found in all forms of obliterating endarteritis, and very frequently are attended by obliteration of the lumen of the vessels. In the syphilitic forms of chronic meningitis wedge-shaped plaques of connective tissue are found in the spinal cord, irregular regions of sclerosis, and a round-cell infiltration all through the white matter of the cord, which occasionally penetrates as deeply as the gray matter. (Fig. 157.) In the syphilitic forms of meningitis these lesions, both of the meninges and the cord, may be continuous over a considerable distance, or they may be disseminated irregularly through either enlargement or through the entire cord.

Symptoms. The course of a case of chronic meningitis differs from that of acute meningitis only in the absence of febrile onset and in the slower development of the various symptoms. The same stiffness in the back and limbs, tendency to cramps, shooting pains, hyperæsthesia, paralyses, anæsthesia, and disturbance in the action of the bladder and rectum, develop slowly, together, or in succession.

Diagnosis. The diagnosis of the condition is usually very easy from the symptoms that are present. Ordinary pains in the back, even when attended by rigidity and by some neuralgic pain and occasional cramps, are not sufficient to establish a diagnosis, as they may be due to neurasthenia, may appear in traumatic neurosis, or may follow slight falls and injuries of the back. Under these circumstances, however, the intensity of the symptoms is much less than in meningitis, and in case of doubt Quincke's puncture will fail to reveal the existence of an increase of cerebro-spinal fluid. Muscular rheumatism may produce a severe pain in the back with more or less tenderness in the muscles and in the spine, but never goes on to develop the other symptoms of meningitis already mentioned. Myelitis, though frequently attended by a complicating meningitis, is not, as a rule, attended by pain of as intense a character, and there are few neuralgic pains and no tendency to a spasm or cramps in the muscles or to rigidity of the back.

FIG. 157.



Syphilitic meningitis. Section from the midthoracic region of the spinal cord, showing the thickening of the membranes and their adhesion to the cord. *a*, *a*, *a*, *a*, *a*. Degeneration of the peripheral parts of the cord. (Spiller.)

Treatment. In the chronic cases a thorough course of inunctions of mercury, followed by iodide of potash in full doses, from twenty to one hundred grains, three times a day, will be of service not only in the syphilitic but also in other cases. Cautery applied to the spine will give relief to many of the pains and to the stiffness. Massage and long-continued warm baths, or spinal douches, or sponging of the spine alternately with hot and cold water, are also of service. The general strength of the patient should be supported, and all forms of good food and tonics should be used freely.

CHAPTER XXIV.

THE DIAGNOSIS AND LOCATION OF BRAIN DISEASES.

THE structure of the brain and the localization of its functions are subjects that have received much attention during the past twenty years. To give even a concise review of them would require too much space in this text-book. Hence the reader is referred to the recent elaborate works of Gordinier,[1] Barker,[2] Edinger,[3] Van Gehuchten,[4] Obersteiner,[5] Dejerine,[6] Ferrier,[7] and the author[8] for the details of anatomy and physiology. In this chapter a study of the symptoms occurring in brain diseases will be presented and an attempt will be made to trace each symptom to its anatomical basis, so that its significance as an index of the pathological state present may be clear.

The symptoms that present themselves in cases of brain disease are broadly divided into two classes, viz., general and local symptoms.

The general symptoms are headache, dizziness, vertigo, discomfort, and general sensations of fulness, pulsation, and pressure in the head; delirium, excitement, depression, stupor, and coma; disturbances of sleep; nausea and vomiting; convulsions; optic neuritis; variations in the rate of pulse, respiration, and temperature; disturbances of secretion of sweat and urine; disturbances in the vasomotor condition, and disturbances of nutrition.

These symptoms may occur in the course of any cerebral affection and indicate an irritation of the brain or an arrest of its functions as a whole. They occur no matter where the disease is located in the brain, and are as common when one locality is affected as when another is invaded. They are important symptoms of cerebral lesion, but they do not indicate its position. They are of great value in determining the variety of disease which is present, and it is from the history of their development in any case that the diagnosis is to be made.

The local symptoms of brain disease, on the other hand, are produced by affections of different parts of the organ, and each symptom shows that a certain particular region is diseased. These symptoms are spasm or paralysis of one or two limbs or of one-half of the body; loss of perception of touch, temperature, pain, or position of one or two limbs or of one-half of the body; loss of visual power or of the power of smell, taste, or hearing; and aphasia, or disturbances in speech. These symptoms may be entirely wanting in some cases of disease when that does not involve the portions of the brain concerned in sensation or in motion. One of them may occur alone if a lesion is of small extent, or they may occur in certain combinations. The important fact, however, in connection with each symptom is that its presence shows the localization of the disease which is present. From the manner of the occurrence of these local symptoms, whether their onset is sudden or gradual, some information can be gained which is an aid in the diagnosis of the nature of the disease, but the character of the local symptom is the same, no matter how different the pathological process. Thus a hemiplegia presents the same features whether the lesion is a hemorrhage, an abscess, or a tumor; but in any of these diseases its appearance indicates that the motor portion of the brain is involved.

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[1] Gordinier. Anatomy of the Nervous System. P. Blakiston's Son & Co., 1899.

[2] Barker. The Nervous System. D. Appleton & Co., 1899.

[3] Edinger. Anatomy of the Central Nervous System. F. A. Davis Co., 1899.

[4] Van Gehuchten. Le Système Nerveux de l'Homme: Van In, Lierre, 1902.

[5] Obersteiner. Anleitung beim Studium des Bauers der Nervören Centralorgane. Vienna, 1901.

[6] Dejerine. Anatomie des Centres Nerveux Paris, 1902.

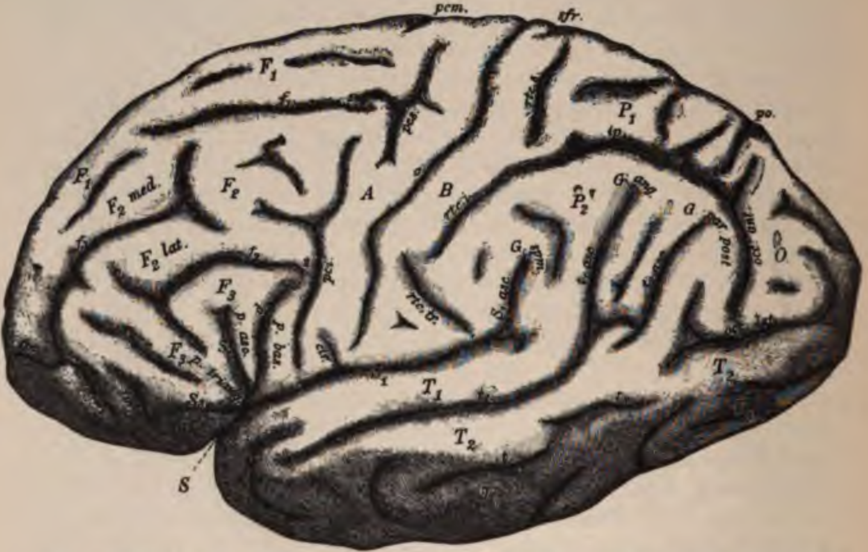
[7] Ferrier in Allbutt's System of Medicine, vol. vii. MacMillan Co., 1900.

[8] Starr. Atlas of Nerve Cells. MacMillan Co., 1896. Familiar Forms of Nervous Disease. Wm. Wood & Co., 1893.

Fig. 158 shows the appearance of the left hemisphere of a highly organized brain. In Fig. 159 a diagram constructed from a series of photographs of the neurones of the cortex is given. This demonstrates the various layers of the cortex and the different kinds of cells in the different layers.

General Symptoms. **Headache** is due to a disturbance of nutrition of the brain. It occurs in a great variety of diseases, being usually more severe in constitutional affections than in diseases of the brain. In both functional and organic diseases of the brain it is a very common symptom, varying in intensity and locality. It may be a dull, continuous pain, with intermissions or remissions and exacerbations, as in the various forms of meningitis and in brain tumor. It may be sudden, sharp, and excruciating, but not long in duration, as in great congestion of the brain, in endarteritis and preceding apoplexy or from the pressure of a tumor. It may be intense for several hours in the afternoon and evening, as in syphilis of the membranes or of the brain. It may take any form in cerebral neurasthenia and hysteria, but never resembles the pain of a neuralgia in its exact limitation to a nerve trunk, or the pain of a migraine in its precise unilateral position and in its regular recurrence in attacks with long periods of freedom. Headache is one of the chief and most severe and agonizing symptoms in pachymeningitis and in meningitis, being either local or diffuse, and is continuous to the end. Headache occurs in mild attacks in the condition of endarteritis which precedes apoplexy, and is likely to be of frequent recurrence on mental or physical exertion. It usually ceases on the occasion of an apoplectic attack, and does not return. Headache rarely develops in cases of cerebral atrophy and maldevelopment; hence idiots, imbeciles, and hemiplegic children do not often suffer from it. It is rarely felt by paretics; hence is not a symptom of meningo-encephalitis. Headache is a marked symptom of brain abscess and of brain tumor, in the former being of sudden onset and severe throughout the short illness, and in the latter being of variable intensity but of almost daily recurrence from beginning to end. Headache is present in thrombosis of the cerebral sinuses. It is a very marked and constant symptom in all forms of brain syphilis. Headache occurs in conditions of anæmia of the brain, being usually felt at the top or back of the head. In the persistence of this symptom and its association with other general and local symptoms of brain disease it is of value in diagnosis. In certain cases headache is always felt at one part of the head—frontal, vertical or occipital, unilateral, in the temple,

FIG. 158.



The outer surface of the left hemisphere. *S.* Sylvian fissure. S_1. Outer portion of the Sylvian fissure; including S_1 *asc*, posterior terminal branch. S_2. Anterior ascending branch. *c.* Central fissure; including *ctr.*, inferior transverse sulcus. *pci.* Inferior precentral sulcus. *pcs.* Superior precentral sulcus. *pcm.* Median precentral sulcus. *rct. i.* Inferior retrocentral sulcus. *rtc. s.* Superior retrocentral sulcus. *rtc. tr.* Transverse retrocentral sulcus. *sfr.* Median marginal fissure. f_1. Superior frontal sulcus. f_2. Inferior frontal sulcus. f_3. Median frontal sulcus. *d.* Diagonal opercular sulcus. *r.* Radiating frontal sulcus. fm_1, fm_2, fm_3. Parts of orbital marginal sulcus. *ip.* Interparietal sulcus. *po.* Inner perpendicular fissure. *occ. ant.* Outer perpendicular, or ape fissure, *occ. lat.* Lateral occipital fissure. t_1. First temporal sulcus, or parallel fissure; including t_1 *asc.*, ascending ramus. t_2. Second temporal sulcus; including t_2 *asc.*, ascending branch. *A.* Anterior central convolution. *B.* Posterior central convolution. F_1. Superior, or first frontal convolution. F_2. Median frontal convolution: including F_2 *med.*, median layer; F_2 *lat.*, lateral layer. F_3. Inferior, or third frontal convolution; including *p. bas.*, basal part of opercular portion; *p. asc.* ascending portion of the opercular portion; *p. triang.*, triangular portion; *p. orb.*, orbital portion. 1. Lateral root of the superior frontal convolution. 2. Lateral root of the median frontal convolution. P_1. Superior parietal lobe. P_2. Inferior parietal lobe; including *G. spm.*, supramarginal convolution; *G. ang.*, angular convolution; *G. par. post.*, posterior parietal convolution. *O.* Occipital lobe. T_1. First temporal, or parallel convolution. T_2. Second temporal convolution. T_3. Third temporal convolution. (Eberstaller.)

or behind the ear. Sometimes such a local pain has been found to be due to disease at the site of pain, viz., to abscess, tumor, especially gumma, or to osteitis, or pachymeningitis; but this is not uniformly the case, and in brain tumors the location of the pain is not a reliable indication of the position of the tumor. The disturbance of the circulation and the venous congestion which lead to an effusion

FIG. 159.

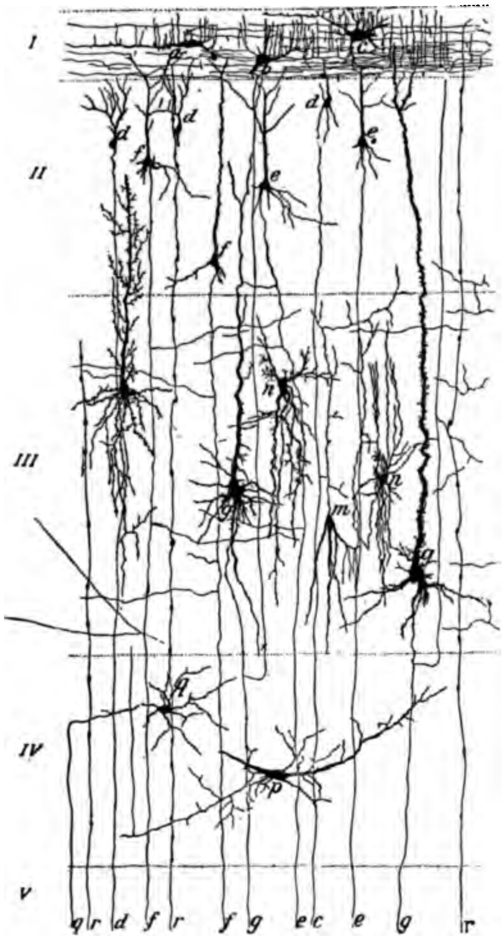


Diagram of the layers of cells of the cerebral cortex. *I.* Superficial layer: *a*, fusiform; *b*, triangular; *c*, polygonal cells of Cajal. *II.* Layer of small pyramid: *d*, smallest, *e*, small, *f*, medium-sized pyramids, with their neuraxones descending to the white matter, giving off collaterals in their course. *III.* Layer of large pyramids: *g*, largest (giant) pyramidal cells; *k*, large pyramidal cell with very numerous dendrites; all pyramidal cells are seen to send long apical processes up to *I.*; *m*, Martinotti cell with descending dendrites and ascending neuraxone; *n*, polygonal cells. *IV.* Deep layer: *p*, fusiform cell; *q*, polygonal cell. *V.* The white matter containing the neuraxones from pyramidal cells *d*, *e*, *f*, *g*, and from cell of the deep layer *p*; *r*, neuroglia fibre. (Starr, Atlas of Nerve Cells.)

of fluid either into the lateral ventricles, into the membranes, or into a cyst in the brain, or to a state of cerebral œdema is a condition which gives rise to most intense and persistent headache, and this is the condition which makes it such a common symptom in brain tumor.

Dizziness and vertigo are common symptoms in brain disease. The origin and varieties of vertigo are discussed in connection with disease of the auditory nerve and its relation to the cerebellum. In diseases of the brain vertigo is usually an intermittent symptom, occurring in attacks. It may consist of a mere sense of swaying, or of the turning of objects about one, or it may be so intense as to force the victim to lie down and to hold on to the bed, the delusion of motion being overwhelming. It leads to an unsteady gait and to staggering. It may lead in extreme cases to forced movements of rotation in an attempt to correct the delusional sense of movement. It is a symptom which appears in many cases of endarteritis prior to apoplexy, and often persists after an apoplectic attack. It occurs in abscess and tumor of the brain, and in these cases may be constant. If persistent and severe in all these cases it may indicate disease in the cerebellum; but, like headache, it is not a reliable local symptom unless it is found in conjunction with other local indications. It occurs in neurasthenia and hysteria as well as in many constitutional conditions. It is undoubtedly an indication of a general disturbance of function and nutrition in those mechanisms of the brain which preside over equilibrium. These are too extensive and located in too many different regions, however, to make vertigo a local sign of disease.

General cerebral sensations of discomfort, of pressure in the head, of fulness, or of irregular pulsation are felt by many patients with functional and organic diseases. They are more commonly noticed in neurasthenia and hysteria and in states of endarteritis that precede apoplexy than in states of brain softening. Anything which increases the intracranial pressure, the existence of a clot, or abscess, or tumor is likely to cause these sensations, and they are invariable with effusion into the ventricles. They are to be considered as of more than casual importance only when associated with other signs of organic diseases.

Mental symptoms of many kinds develop in the course of brain disease and are always due to a disturbance of cerebral function. A state of mental excitement may indicate an irritation of the brain cortex, and this may become delirium when the power of voluntary control of thought is impaired. This occurs in abnormal constitutional states and fever and in meningitis much more frequently than in gross lesions of the brain, though after an apoplexy, or in the course of an abscess, or thrombosis of the lateral sinus, or in syphilis of the brain, delirium at night is not uncommon. Depression of brain activity, a sense of inability to think or reason, a true slowness of the association processes, and an impairment of judgment and of self-control are symptoms of cerebral lesions found in all varieties of disease, both diffuse and localized. The lack of self-control manifests itself by an emotional state in which the patient shows signs of amusement or of distress on slight cause, and is uncontrollable in temper and action. All these forms of mental disturbance may develop in any kind of disease of the meninges or of the brain. They are particularly prominent in cases of affection of the frontal lobes, which, as is well known, are the supposed site of the higher mental faculties, of co-ordinated ideas, of powers of attention, of reasoning, of judgment, and of self-control. Defects of development in these lobes are followed by an inability to acquire the higher ideas which distinguish man from animals, and hence lead to weak-mindedness and idiocy. Therefore, such mental symptoms may be considered as local symptoms of frontal lesion in many cases. They occur with diseases within the frontal lobes as well as in cortical affections, and also in lesions of the corpus callosum, whose fibres associate the action of the hemispheres. They are particularly common in lesions of the so-called association areas of the cortex. They are less marked in local disease of the brain that is confined to the motor and sensory regions. Yet they do occur in general diffuse affections, such as paresis and multiple cerebral softening, and hence must be regarded as general symptoms rather than as purely local. The various types of insanity rarely develop in cases of gross cerebral disease, but dementia is the terminal mental state in many cases of extensive destruction of brain tissue.

Insomnia is a common symptom of all forms of intracranial disease that cause an irritation of the brain. It accompanies cerebral congestion and anæmia, but is not due to either exclusively. It is always present in meningitis. It often attends gross lesions, especially those that increase the intracranial pressure. After an apoplexy patients usually sleep badly, are restless, and wake easily. Sometimes the sleep is disturbed by dreams. Insomnia is an early sign of paresis. Grinding of the teeth in sleep is a sign of irritation of the base of the brain. It is most common in meningitis and in basal tumors. The opposite condition of drowsiness is observed in many chronic cases of brain tumor, in cases of dementia, in cases of extensive softening, and in cases of paresis. Narcolepsy or sleeping sickness, in which patients fall asleep at unseemly times and places, and cannot be aroused, is a functional state not found in connection with gross lesions.

Stupor and coma develop after a sudden shock to the brain, such as occurs in concussion of the head, or hemorrhage in the brain, or with a rapid alteration of circulation in the cerebral vessels, as in the extreme congestion of sunstroke, or the anæmia of hemorrhage, or in a condition of sudden œdema of the brain such as follows thrombosis and embolism. In these conditions coma comes on suddenly. It occurs in apoplexy when the clot is large, when it lies near the lateral ventricle, or when a large vessel is obstructed and a large area of the cortex is deprived of blood. In any disease that leads to an increase of the intracranial pressure stupor and coma may develop slowly. Thus in the various forms of meningitis and sinus disease, in encephalitis, in brain abscess and tumor, in syphilitic exudations, in ventricular effusions coma comes on after the disease has been in progress some time. Coma usually succeeds a convulsion. Many poisons, either of external or internal origin, are capable of causing coma, and hence it is not to be considered as a symptom of brain disease unless associated with other signs. It has no local significance, but when it occurs in the course of a cerebral affection it is a sign of great shock or extensive disease of the brain, and hence is of bad prognostic importance.

Nausea and vomiting are only to be considered as cerebral in origin when all forms of disturbance of the stomach and other organs can be excluded. Nausea is a very rare symptom of brain disease, but occasionally precedes vomiting of cerebral origin. Vomiting is a sign of irritation of the cerebral tissue dependent upon irregularities of the circulation or upon an increase of intracranial pressure. Thus it occurs in endarteritis, at the onset of apoplexy, in the condition of brain abscess and brain tumor, and is a very common symptom in every form of meningitis. Cerebral vomiting often comes unexpectedly, not being preceded by nausea or attended by retching. It is due to a sudden convulsive contraction of the stomach, which expels its contents violently; hence it is called projectile vomiting. It is more common in affections of the base of the brain or of the cerebellum than of other parts, and is then probably due to local irritation of the pneumogastric nerve. But it is not to be regarded as a local symptom, since it occurs in diseases in any locality.

Convulsions of a general kind affecting the entire body and attended by loss of consciousness are either due to a toxic agent in the blood which irritates the brain or to some source of irritation affecting the organ directly. Alcohol, lead, uræmia, high fever in any disease, and many drugs may cause convulsions. Meningitis of any type, encephalitis, paresis, abscess, tumor of the brain, and syphilis in any form may produce them. Epilepsy is characterized by convulsions the cause of which is unknown, and in hysteria convulsions may develop. Intermittent peripheral irritation, frequently repeated and transmitted inward to the brain may, in a hypersensitive organism, cause sufficient cumulated central irritation to produce a general convulsion. A convulsion may consist of a series of alternate contractions and relaxations of the muscles of the body, termed clonic convulsions, or may be a tense rigid contraction of many muscles together, termed a tonic convulsion. In epilepsy the tonic stage precedes the clonic. The two are usually associated in a general convulsion, alternately. The convulsion may begin with a cry, which is merely the result of a sudden spasm of the muscles of expiration, may lead to biting of the tongue and frothing at the mouth, the saliva being churned up by combined motions of jaw and respiration, may be accompanied by an emptying of the stomach, bowels, and bladder, if these are full, and is usually followed by a complete relaxation of the tired muscles and a state of stupor or coma lasting a variable time, in proportion to the severity and length of the convulsion. The convulsion may last from two minutes to ten or even twenty. It may be repeated every few minutes for hours, or there may be but one attack.

Physiological experiment points to the existence of a centre in the pons Varolii the irritation of which produces a general convulsion, but many clinical facts seem equally to prove that irritation of the cortex of the brain, at first local, may become general, and thus cause a convulsion. A convulsion is not a local symptom, as disease in any part of the brain may bring it on. Gowers[1] conceives a condition of the motor elements of the brain as one quite comparable to the condition of a particle of dynamite or gun-cotton, ready at any time to give a manifestation of its stored-up energy by a sudden discharge. In reflex acts a slight irritation is capable of exciting a single response. In voluntary acts a conscious effort causes a regulated and maintained discharge of energy. In a convulsion there is an involuntary, tumultuous, and irregular discharge which continues to the point of exhaustion. Others have thought that a convulsion was rather an evidence of paralysis of a higher set of controlling or inhibitory centres whose constant action is exerted in repressing the instinctive tendency of motor elements to act. These hypotheses have much interest but little practical importance.

Optic neuritis is a general symptom indicating an increase of intracranial pressure. It is fully discussed in Chapter XXXIV. It may occur in any case of brain disease that is attended by effusion of blood or serum in the membranes or brain, in ventricular effusions, in states of extreme congestion, in brain abscess, and in brain tumor. It has no localizing value as a symptom, but is more commonly found in affections of the base of the brain, of the basal ganglia, and of the posterior cranial fossa. It rarely develops suddenly. It may be well marked without causing blindness, but it usually results in a dimness of vision and, if extreme, in a loss of sight.

Variations in the pulse may attend cerebral disease. A slowly increasing intracranial pressure at first causes a slow pulse, but when it is sudden or extreme the pulse may be rapid. Thus a large hemorrhage causes a rapid pulse, a tumor causes a slow pulse during its course and a rapid pulse at the end. Variations in the cerebral circulation, such as attend endarteritis and thrombosis or embolism, cause sometimes a rapid, sometimes a slow, action of the heart. Irritation of the base of the brain, such as occurs in meningitis, always produces an irregular and frequently an intermittent pulse.

Variations in the rate of respiration may be produced by intracranial disease, but are less common than changes in pulse rate. A sudden increase of intracranial pressure, such as occurs in hemorrhage or serous effusions of considerable size, will produce an irregular respiration consisting of rapid shallow breathing, gradually growing slower, followed by an entire arrest and then by a long inspiration again, followed by rapid breathing. This is termed Cheyne-Stokes respiration, and is a sign of unfavorable prognostic import. A permanent slowing or hastening of respiration has not been observed in organic disease, but may occur in hysteria.

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[1] The Dynamics of Life. London, 1897.

Variations in temperature might be expected as a frequent symptom, since the regulation of body temperature is controlled by cerebral centres which are located by Ott in the basal ganglia. As a matter of fact, they rarely appear in cerebral disease excepting when they are due to general constitutional disturbances. A sudden fall of temperature is observed after a large cerebral hemorrhage, and is succeeded by a gradual rise which, in occasional fatal cases, attains a very high degree. Sometimes, after an apoplexy from any cause, the temperature on the paralyzed side becomes one or two degrees higher than on the other side. It is very rare, however, for a brain tumor to cause fever, no matter what its location or the region of the brain that it irritates or destroys.

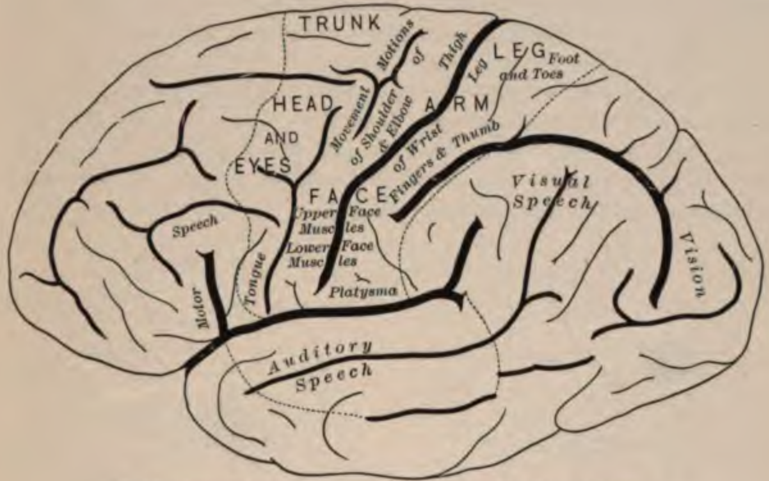
The secretion of urine is under the control of a nervous mechanism in the medulla, and local lesions in the vicinity of the pneumogastric nuclei have been known to cause diabetes insipidus and mellitus. Some irritation of these nuclei may occur from disease located elsewhere in the cranium. Thus an apoplexy, no matter what its cause or location, is usually followed at once by an increased excretion of urine, and this may contain albumin and sugar for a few days. Brain tumors may also cause this symptom from time to time in their course. Hence polyuria or glycosuria must be regarded as general rather than as local symptoms. They are not infrequent symptoms in hysteria, in which disease a suspension of renal activity may also occur as a nervous symptom.

The vasomotor condition of the body is also regulated by centres in the medulla that any disease at any point in the brain may irritate or may affect. Hence a unilateral venous congestion, with relaxation of the vascular walls, undue sweating, and local œdema may attend cerebral hemorrhage or softening. It is also a symptom of meningitis and occasionally of tumor.

Emaciation. In some diseases of the brain patients become rapidly emaciated. This happens in many forms of meningitis, especially in the posterior cranial fossa, and in disease of the cerebellum. I have also seen it in tumors of the corpus callosum and of the frontal lobes. It appears to be irrespective of the amount of food or of the digestive capacity and without relation to any known factor. It must, therefore, be due to some affection of those nervous centres which preside over the mechanism of metabolism. Their position in the brain is unknown.

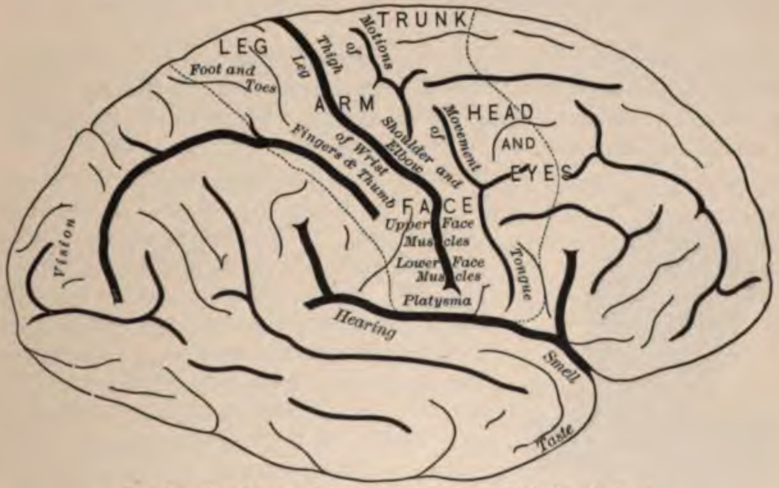
Local Symptoms. **Convulsive movements** limited to a portion of the body and, when extending to other parts, following a definite order of progress, are symptoms of irritation in the cortical motor area of the brain. Such irritation must originate in the cortical cells, as spasms rarely occur from subcortical lesions. Fig. 160 shows the limits of this area in the left hemisphere and its subdivisions. It lies on both sides of the fissure of Rolando and includes adjacent portions of the cortex at the posterior end of the three frontal convolutions and at the anterior part of the parietal lobules. It also includes the cortex of the paracentral lobule on the median surface of the brain. In the right hemisphere (Fig. 161) its area is a little

FIG. 160.



The functional areas of the cerebral cortex. Left hemisphere.

FIG. 161.

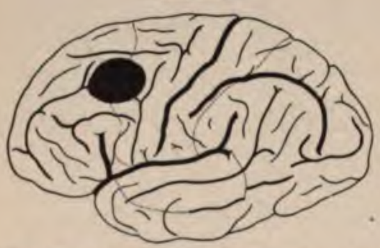


The functional areas of the cerebral cortex. Right hemisphere.

smaller in right-handed persons, for each hemisphere controls the movements of the opposite side of the body.

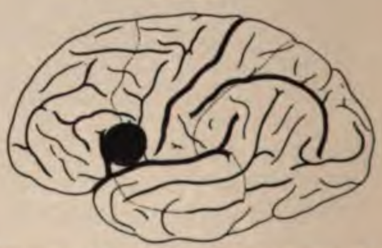
Limited convulsive movements are termed *cortical spasms* or *Jacksonian epilepsy*, after Hughlings Jackson, who described them in 1861. Spasmodic turning of the head and eyes, or of the eyes alone to the right, indicates irritation of the posterior part of the second frontal convolution in the left hemisphere. Such movements to the left indicate irritation in the same area of the right hemisphere. The head turns in a series of jerks, and the eyes have a conjugate lateral jerky movement—nystagmus—and these go on until the patient looks over the shoulder. In Fig. 162 the situation of a tumor is shown which caused this form of cortical spasm as the uniform commencement of Jacksonian epilepsy.

FIG. 162.



Situation of tumor causing spasm of head and eyes to the right; successfully removed.

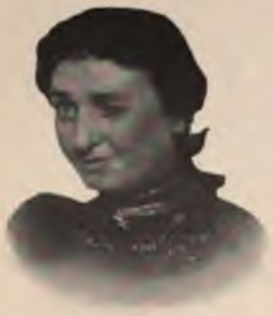
FIG. 163.



Situation of tumor causing spasm of right side of face and tongue; also motor aphasia.

Spasmodic twitching of the facial muscles on one side indicates an irritation in the lower third of the motor area at the foot of the central convolutions. Such twitching may begin in the forehead, or in closure of the eye, or in the muscles of the nose, or about the mouth, or in the chin, or the cheek may be drawn down by the platysma, or the tongue may twitch, or spasmodic noises may be made by a laryngeal spasm. In Fig. 160 the various subdivisions of the facial area are shown, each portion controlling the muscles named. Wherever, in the area, the irritation begins it usually spreads to the entire region, so that the entire side of the face is thrown into a spasm. The spasm is clonic, the rate of movement being six or eight contractions to the second, and the spasms may continue for several minutes. Fig. 163 shows the situation of a tumor which caused such localized convulsions as the uniform beginning of a Jacksonian epilepsy, and Fig. 164 shows a patient in the act of having such a spasm of the face.

FIG. 164.

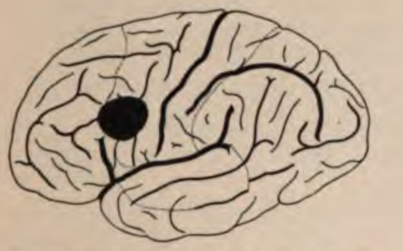


Localized spasm of the face in a patient suffering from Jacksonian epilepsy.

Spasms of the face often extend to the eyes and head, and *vice versa*, as the irritation spreads outward from either area to the adjacent one. This occurred in the case shown in Fig. 165, where the upper facial muscles were involved after the head and eyes had turned to the right.

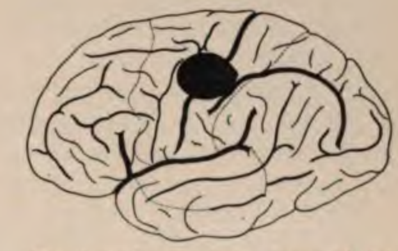
Convulsive movements limited to the upper extremity show an irritation in the middle third of the motor area. The spasm may begin with a drawing upward of the shoulder and turning downward of the head, as in the case shown in Fig. 166, or by any movements of the arm at the shoulder-joint, or by movements of the elbow of flexion and extension, or by movements of the wrist and fingers and thumb. Or the spasm may begin in the hand and ascend the arm, involving all the muscles in the reverse order named. Usually the hand is closed, the wrist flexed and pronated, the elbow flexed, and the arm adducted when the spasm is at its height, and the limb trembles violently in the convulsion. The flexors thus overcome the extensors in the conflict between both in the spasm. Occasionally large movements at the joints are made, and the hand makes wide excursions, as in one case of mine, following a cortical hemorrhage, where the arm was flung about like a flail. It is important to notice the order of extension of the spasm in the arm, as this gives a clue to the point on the cortex where the irritation begins. The subdivisions of the motor area for the arm are shown on Fig. 161. In Fig. 167 the location is shown of a tumor removed successfully from the cortex. In this case the patient suffered from many Jacksonian spasms of the upper extremity which always began with a twitching of the thumb and first finger, then a closure of the hand, and then extended up the arm. Spasms beginning in the upper extremity may extend to the face, or to the head and eyes, or to the trunk and leg. Reference to Fig. 161 demonstrates that this extension merely indicates a radiation outward of the initial irritation to adjacent areas. And it is evident that the order of extension of the spasm gives a clue to the position of the point of initial irritation. Thus in the case shown in Fig. 166 the spasm extended to the face from

FIG. 165.



Situation of tumor causing cortical spasm and motor aphasia. Head and eyes turned to the right, then forehead and cheek were drawn to the right.

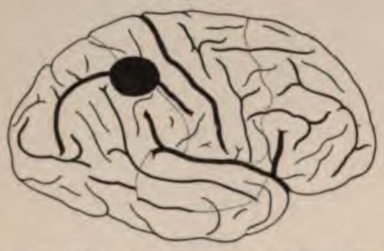
FIG. 166.



Situation of cyst causing spasm of right shoulder, extending down the arm and to the fingers. Cyst removed.

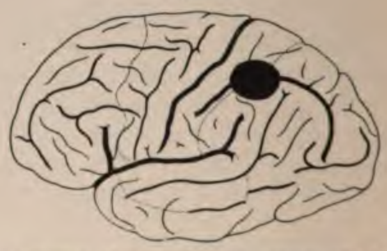
the arm. In case shown in Fig. 168, where the lesion was a cortical tumor, the spasm extended from the arm to the leg before it reached the face. When a spasm first affects the shoulder, the trunk, and head, and eyes are usually thrown into convulsion before the leg or face. It is evident from a study of a large number of such cases of Jacksonian epilepsy that the order of extension of the spasm is not a matter of accident, but is governed by the spread of irritation from one cortical region to the adjacent region. One has to think of such a spread of irritation as resembling the little waves on a lake which extend from the place where a stone strikes the surface in concentric but ever-widening circles.

FIG. 167.



Situation of tumor causing spasm of left fingers and thumb, then of wrist, elbow, and shoulder. Tumor removed.

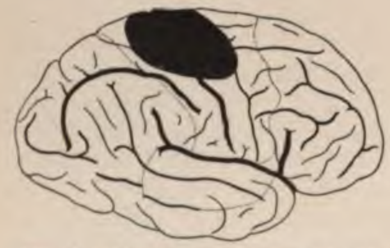
FIG. 168.



Situation of tumor causing spasm of the right thumb, fingers, and arm, then of foot and leg, finally of face. Tumor removed.

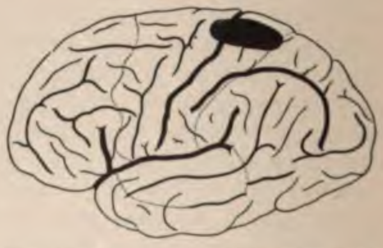
Convulsive motions limited to the trunk are usually of the nature of bending of the body toward one side with a drawing up of the thigh. They are not often observed alone, but commonly occur as the spasm extends from the shoulder to the leg or from the leg to the shoulder.

FIG. 169.



Situation of tumor causing spasm beginning in the left thigh, extending to the body, arm, and face. Removal impossible.

FIG. 170.



Situation of meningeal thickening and adhesion after trauma, causing spasm of leg, beginning in knee, and extending to foot and hip.

Convulsive motions limited to the lower extremity may begin in the thigh, as in the case shown in Fig. 169, and extend down the leg, finally involving the foot and toes, or they may begin with a twitching of the toes which extends to the ankle and knee, and finally to the hip, as in the case shown in Fig. 170. The lower extremity is usually extended in a Jacksonian attack, is held rigid, and trembles, the extensors overcoming the flexors in their action. When a spasm begins in the leg it extends to the arm or trunk before it reaches the face, and usually involves the head and eyes before it affects the lower facial muscles or the tongue.

Local spasms are always attended by a sensation of tingling in the part that is convulsed. Sometimes the tingling sensation precedes the actual movement by some seconds or even minutes. This tingling has been named by Seguin the signal of an attack. It is an interesting fact that if a strong irritation of the skin is made in or near to where the tingling is felt as soon as the tingling begins, the spasm may be averted. Thus I have a patient who wears a strap about his wrist and who pulls it tight as soon as he feels a tingling in his fingers, in this manner preventing the occurrence of a Jacksonian spasm of the hand and arm, that is sure to follow when he omits the constriction of the wrist. Such a tingling sensation is more commonly felt in the fingers and hand, in the toes and foot, or in the face than it is upon the trunk or high up on the limbs, probably because the sensibility of these parts is more highly evolved and keener than in the others. Or, to state it differently, the sensations in the periphery of the limbs are more accurately localized and represented in the cortex. The occurrence of these sensations with the local spasm is an argument for the coincidence of sensory and motor functions in the cortex.

Local spasms are always followed by a condition of voluntary weakness which may amount to an actual flaccid paralysis in the muscles which have been convulsed. These muscles cannot be moved for some minutes or even hours, but, finally, unless the original lesion is a destructive one, power returns. The paralysis lasts longer in the muscles in which the spasm begins than in the others. Hence the subsidence of the paralysis presents an order of succession in the limbs which is just the reverse of the order of invasion in the spasm. Thus in the case shown in Fig. 168 the patient was quite hemiplegic for an hour after each attack, then the leg began to recover, then the face, then the upper arm, and finally the power returned in the hand and fingers. When, in the growth of the tumor, however, she began to be permanently paralyzed, the paralysis extended in the reverse order, following the order of extension of the spasm. Thus the order of extension of a spasm and of a permanent paralysis is just reversed in the order of recovery in a temporary paralysis. The tumor was removed two years ago, and she is now perfectly well.

All these facts are of great importance as an indication of the extent and location of small cortical lesions in the brain, and are the main guides to the surgeon in trephining.

Local spasms do not invariably indicate a primary lesion in and a direct irritation of the motor region. Such irritation may be indirect; that is to say, an irritation may start from a point on the cortex outside the motor area and, as it extends, finally reach that area, and then set up a spasm. The spasm will begin in the limb the motor area of which is the part reached by the irritation, and then if the spasm extends it may follow the regular order. Sometimes an irritation starting in the occipital or parietal region and extending forward reaches the motor area of the fingers and toes simultaneously. Then the spasm will begin in the hand and foot together, extending up both limbs at once. This may, therefore, be a clinical fact of importance in showing that the lesion lies outside the motor zone. Thus in Fig. 167 the location of a tumor is shown which caused Jacksonian attacks which sometimes began in the toes, sometimes in the fingers, and always involved both extremities in part before either was entirely convulsed. In such cases other sensory or aphasic, mental symptoms may precede the attack. Much care must, therefore, be taken in watching the exact characteristics of Jacksonian epilepsy.

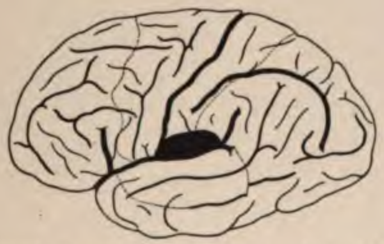
Another fact of interest is that the movements and spasms due to a cortical irritation always resemble voluntary movements. Jackson says: " The convulsion is a brutal development of a man's own movements. A severe fit is nothing more than a sudden excessive and temporary contention of many of the patient's familiar motions, such as winking, articulating, singing, manipulating," etc. They are never contractions of single muscles or of groups of muscles which may have a nerve supply from one nerve alone, or which may have a relation in their spinal representation. Thus cortical spasms can always be differentiated from spasms due to irritation of a nerve trunk or of a spinal nerve root, or of the motor mechanism of the spinal cord. They are not like reflex acts. The cortical movements can always be voluntarily imitated. They are always apparently for a purpose and show a certain amount of co-ordination and adaptation to an end. Cortical acts have always been acquired by a process of education which can be seen in progress by watching an infant. The act, when finally learned after many attempts and repetitions, leaves a memory which has, as a physical basis, an organized and inter-related group of cells in the cortex. Irritate this group, and the act is performed. Thus cortical movements may be started by electrical applications through needles to the cortex, and in surgical operations for Jacksonian epilepsy it is well to explore the cortex with a faradic current, to elicit movements until those are produced which exactly resemble the spasm of the disease, and then to extirpate the area whose exact function is thus established. The localized spasms of cortical disease are rarely, if ever, successfully imitated either by malingerers or by hysterics. A hemispasm sometimes occurs in hysteria, but it usually begins in both limbs at once, and if it extends from one limb to the other, or if it extends along a single limb, it rarely preserves the exact order of extension so uniformly observed in local lesions. Hence it is not difficult to distinguish cortical epilepsy from hysterical attacks of a convulsive nature.

Localized convulsions do not induce a loss of consciousness. Localized convulsions may extend from one part to another until the entire side is in a state of spasm. They may then extend to the other side of the body, setting up general convulsions, which may be accompanied by loss of consciousness. Whether, in such cases, the irritation is conveyed to a convulsive centre in the pons Varolii or to the motor cortex of the opposite hemisphere by way of the commissural fibres is not yet determined.

Irritation in the cortex does not necessarily lead to convulsions. The Jacksonian attack may be exclusively sensory, leading to hallucinations, or when in the speech areas, leading to temporary attacks of aphasia, or to mental states of disturbance of consciousness, or of the will, or of the memory. But any of these forms of attack may go on to a localized spasm, and a localized spasm may in its turn produce subsequently one of these peculiar states. They will be more fully considered when sensory and aphasic symptoms are discussed.

Paralysis from Cortical Disease. Paralysis is a symptom of local lesion in the motor area of the brain or in the tract from it to the spinal cord. This paralysis is one of voluntary motion, the reflex and automatic acts presided over by the spinal and subcortical basal centres being in no way interfered with. The character of the paralysis depends largely upon the location of the disease and upon its extent.

FIG. 171.



Situation of focus of hemorrhagic encephalitis, causing paralysis of the tongue and lips. (Dana.)

Paralysis of motion of the face, arm, or leg, of the head and eyes, or trunk, may be due to cortical disease when the motor area controlling their movements is affected. From the extent of the paralysis it is possible to arrive at a conclusion regarding the extent of the lesion in the cortex. Lesions of the cortex, as a rule, are not very extensive. Thus cortical hemorrhages are rarely very large, softened areas from embolism or thrombosis in the cortex are limited as the collateral circulation is good; an abscess or a tumor in the cortex is at first small. Therefore, as a rule, from cortical disease we get a paralysis of limited extent, the face alone, or arm alone, or these together being paralyzed; or the leg alone, or arm and leg together being affected. This is termed monoplegia or associated monoplegiæ. Since each hemisphere of the brain controls the opposite side of the body chiefly, the paralysis from a lesion in one hemisphere is limited to the opposite side of the body. Paralysis of one side is termed hemiplegia. In cases where both hemispheres are involved we may have double hemiplegia or diplegia; but this is a rare condition. As a matter of fact, each hemisphere is connected by the motor tract with both halves of the spinal cord, as we shall presently see, and therefore in every case of hemiplegia the unparalyzed side is really a little weaker than before the attack. There are rare cases where the hemiplegia has been on the side of the lesion. These are, however, explained by an absence of a decussation of the motor tracts in their course. But the separation of various motor areas from one another in the cortex, and the usual limitations of lesions to small regions in the cortex, makes hemiplegia from cortical disease rare. The usual result of a cortical lesion is a monoplegia.

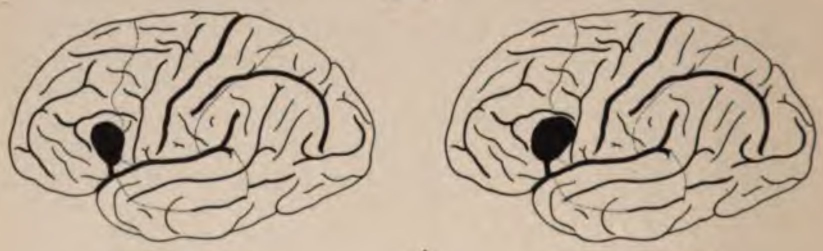
The Characteristics of Voluntary Action. The paralysis, as already stated, is one of voluntary motion that has been acquired by practice. If we study voluntary acts as they are performed we find that each act involves a succession of movements by different joints, each movement being produced by the contraction of a number of different muscles. The act of lifting a feather or a heavy weight involves the same muscles, but there is a great difference in the order, sequence, and relative force expended in the two acts. In both the entire muscular system of the arm is called into play, but in each there are some muscles whose action is greater in degree and precedent in time than in the other. The motor centres of the cortex govern acts rather than muscles. It is the act of grasping, of lifting, of opening the hand, of pointing, etc., that is produced by cortical activity rather than mere contraction of flexors and extensors of the fingers. Now any such act involves the co-ordinated movement of many muscles for its production. We must think, therefore, of the motor area of the hand, for example, as containing an enormous number of centres of action arranged in groups, so that it cannot be divided by sharp lines into flexor and extensor centres, since flexor and extensor action occur simultaneously in many acts. If the motor area be divided at all it must be into regions for various acts, each having its predominant feature. Hence in subdividing the arm area into regions for shoulder, elbow, wrist, and fingers it is intended to separate actions whose special feature is the movement of the joint named. In each division all the joints may be represented in part, but in different degrees. Thus the lifting of the arm above the head is primarily a shoulder movement, but involves some action in all the other joints, since the forearm and fingers do not hang limp. And a firm grasp, though primarily a flexor action of the fingers, involves some act of support by all the muscles of the arm. In the case shown in Fig. 166 the shoulder motions were more defective than those of the hand. In the case shown in Fig. 168 the hand motions were impossible and the shoulder was only partially paralyzed.

As the pianist, by striking the keys in different combination, produces an unnumbered variety of harmonies from a small number of strings, so the cortex, by exciting the spinal neurones, and through them the muscles, attains a countless variety of movements. The vibration of a single string gives sound but no music, and the contraction of a single muscle gives motion but no act. The loss of a single string causes discord, but does not stop the sounds, for other strings still vibrate. The paralysis of a muscle impairs motion, but does not stop the act. The strings lie silent unless the keys are struck. The muscles are not called into action unless a cortical impulse reaches them. Thus we must regard the cortical motor centres as different in their action from the spinal motor neurones. They are the storehouses of memories of movements, and are capable of reproducing these movements which they have acquired. Skill in any complex act, like that of the musician, is only gained by numberless repetitions. Watch the process of learning to talk or to write in a child, and it will be evident that countless futile attempts precede success. Such attempts are really simultaneous acts in many centres, causing inco-ordinated movements, but little by little one is subordinated to another in vigor, and one is made to precede another in time until an order is reached and an accuracy is attained, and this by repetition becomes a fixed combination, as easy for the cortex as is the motion producing the harmony for the pianist. That the simplest acts are first acquired is to be expected, but there is no limit to the skill possible and no limit to the variety of complex movements which may be learned. Every artisan, every musician, every dancer, has a peculiar individual store of motor memories. Some individuals possess a greater variety of them than others. Hence the motor zone on the cortex is of different extent in different persons, each newly acquired set of movements increasing its area.

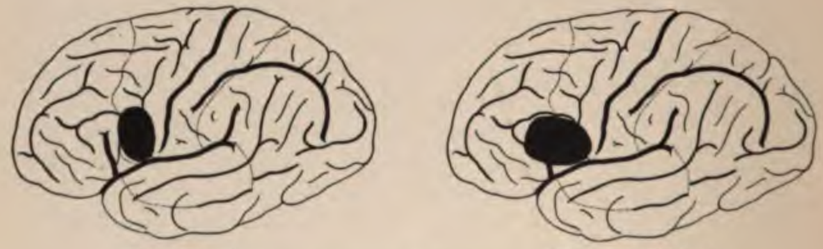
The Motor Centres of Speech. There are some highly complex motor acts which seem to have a special region assigned to them. The act of speech and the act of writing are such, and these are located in the posterior part of the third and second frontal convolutions, respectively. The power to speak and to write are lost when these regions are destroyed by disease. Yet such a destruction does not necessarily involve a paralysis of the muscles which carry out these acts; it does not even suspend the voluntary movements in these muscles, which can be well performed, but not in the combination needed to prduce speech or writing.

The loss of the power of speech is termed aphasia. There are two types of aphasia, which are recognized as motor and sensory. In motor aphasia the act of talking or writing is lost. In sensory aphasia the understanding of words heard or seen is lost. The centres which preside over the emission of language are highly complex motor centres which appear to utilize the secondary motor centres in the facial area, setting them to work in varying combinations. When the facial centres are paralyzed attempts at speech may fail from imperfect pronunciation or articulation. This is termed anarthria, and is attended by paralysis of the tongue and lips, as in the case of Dana, shown in Fig. 171. When the motor speech centres are paralyzed no attempt can be initiated, and hence words cannot be produced either by voice or hand. In Fig. 172 a number of cases are shown where the lesion caused motor aphasia. The state of motor aphasia is not attended by any loss of comprehension of language. The understanding is good, and the patient knows what he wants to say but cannot find the words. Nor can he repeat words after another. The motor combination appears to be lost. The condition may be general or partial. Some words may be said while others cannot, and sometimes words are possible, but their combination in sentences is impossible. Writing is usually lost with speech.

Fig. 172.



Tumor. (Starr.) Tumor. (Dejerine.)



Clot. (Starr.) Clot. (Hotchkiss.)

Situation of lesions causing motor aphasia.

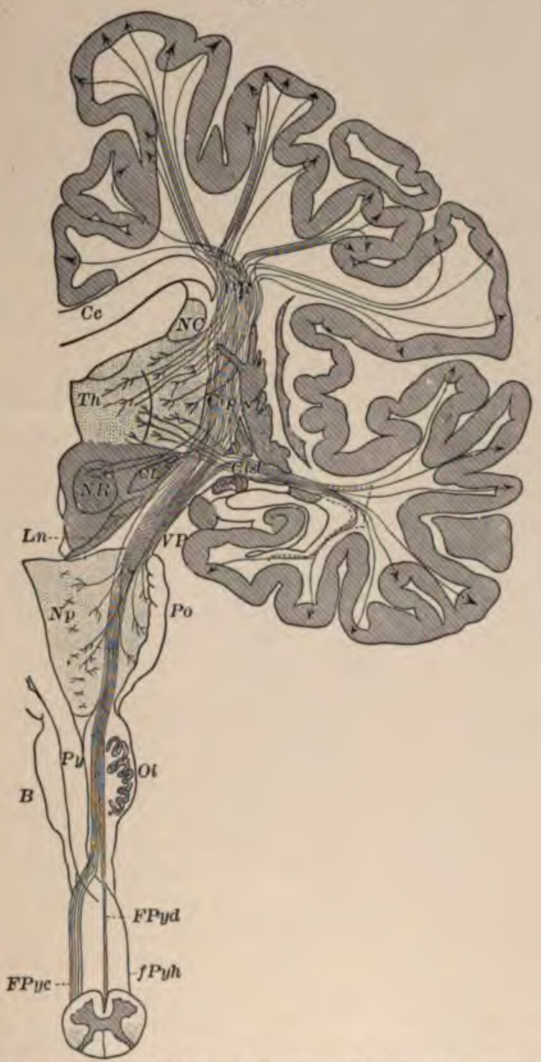
Here, then, is another proof that the cortical motor acts are not simple muscular motions, but are highly complex combinations. It is as if an electric key by one pressure started a whole mass of machinery whose different parts were independent and wholly unlike in their activities.

The paralysis from cortical disease is therefore a suspension of the power of highly complex actions, and when it is due to a destruction of the cortical centres it is attended by a loss of the memories of effort essential to those acts.

Paralysis from Subcortical Disease. Paralysis may also be due to an interruption in the tract which conveys these cortical impulses to the motor centres in the base of the brain and spinal cord. This tract is called the motor tract. Its course is shown in Fig. 173.

It arises from the under surface of the cortex of the posterior part of the third frontal convolution, the two central convolutions, and the paracentral lobule, and passes out of the base through the middle

FIG. 173.



Scheme of the projection fibres of the cerebral cortex. *Cc.* Corpus callosum. *Cr.* Internal capsule, into which fibres pass from the cortex above the Sylvian fissure. *NC.* Caudate nucleus. *NL.* Lenticular nucleus. *Th.* Optic thalamus, in which many fibres from all parts of the cortex end. *Cip.* Posterior division of the internal capsule, corresponding to the sensory tract in Fig. 178. *Cisl.* Sublenticular part of the internal capsule, containing fibres of auditory tract and fibres to the thalamus from the cortex below the Sylvian fissure. *CL.* Luys' body. *NR.* Red nucleus of tegmentum. *Ln.* Locus niger. *VP.* Tract from capsule to pons. *Po.* Pons. *NP.* Gray matter of pons. *Oi.* Olive. *Py.* Pyramidal tract in medulla, passing to *FPyd*, anterior median column of cord, and to *FPyc*, lateral column of cord of opposite side, and to *fPyh*, lateral column of cord of same side. *B.* Medulla. (Dejerine, Anatomie des Centres Nerveux, vol. ii. p. 2.)

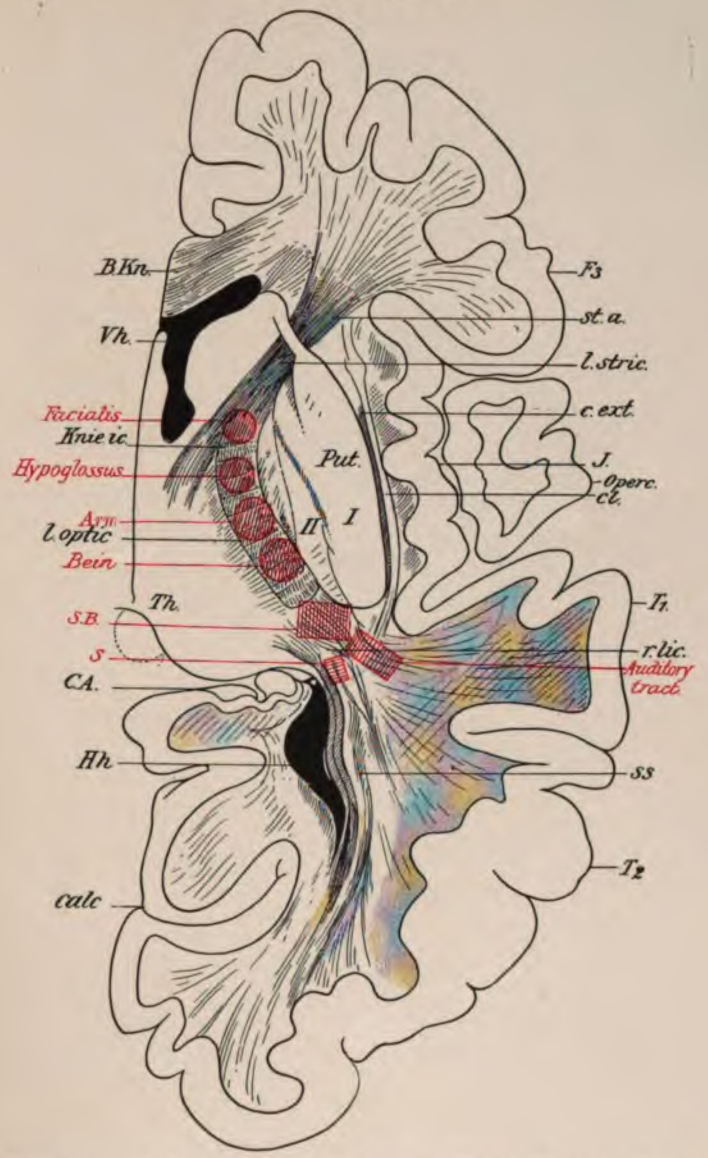
third of the crus cerebri. Its fibres gather together at the middle portion of the upper surface of the internal capsule; those from the lower parts of the cortex passing straight inward, those from the upper parts curving outward and downward to pass around the side of the lateral ventricle. Thus, within the centrum ovale these fibres, if looked at from in front, appear like the sticks of a fan, and, like those sticks, their relative position is altered in the point of junction, where those passing inward from the lowest part of the cortex lie in front of those that pass downward from its upper part. Thus in the capsule the order from before backward is, first, the fibres conveying speech impulses to the pons and medulla; second, the fibres conveying facial-motor impulses to the pons; third, the fibres destined to the arm centres of the cord; fourth, the fibres transmitting impulses to the leg centres in the cord. The fibres conveying impulses to the muscles of the trunk probably lie behind those to the leg. Plate XVIII. shows the relative positions of these fibres in the internal capsule.

From the anterior half of the posterior division of the capsule this tract passes through the middle third of each crus (where the fibres controlling the movements of the eyes are given off), through the pons (Fig. 174) (where the division to the facial nucleus crosses to the opposite side and ends), and thence by way of the pyramids of the medulla to the crossed pyramidal and direct anterior median columns of the spinal cord. It is evident, however, that the concentration of this tract is much greater in the capsule than in the centrum ovale, where the individual fibres are scattered among the other systems and occupy but a small area from before backward.

The character of paralysis occurring from lesions in this tract is also shown in Fig. 174. Four lesions are there shown: one in the centrum ovale near the cortex, one in the internal capsule, one in the crus, and one in the pons. It is evident that the lesion in the centrum ovale affects one set of fibres only, those from the arm centre, and produces, therefore, a monoplegia only. A lesion of the centrum ovale would have to be very extensive to involve the fibres from the face, arm, and leg centres together. But a small lesion in the internal capsule cuts all these fibres where they pass in a condensed tract, and hence produces a hemiplegia, face, arm, and leg being all paralyzed on the opposite side. It is very rare to get a monoplegia from a lesion in the capsule. Any clot larger than the size of a pea destroys the entire motor tract here. The vast majority of cases of hemiplegia are due to a lesion of the internal capsule. And the figure shows that it is only in their passage through the capsule, the crus, and the upper part of the pons that these fibres can be affected together by a single small lesion.

A lesion in the crus cerebri causes hemiplegia; but since the optic tract crosses the crus, and the third nerve to the ocular muscles comes out from its inner surface it is hardly possible for both of these nerves to escape in a lesion at this point. Hemiplegia associated with

PLATE XVIII.



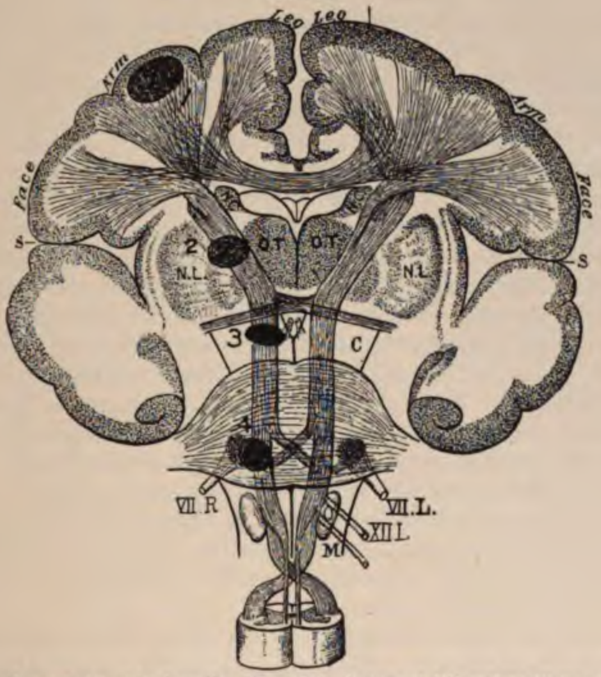
Horizontal Section through the Right Hemisphere.
(After von Monakow.)

The important parts of the internal capsule are colored red. *B. Kn*, knee of corpus callosum; *Vh*, anterior horn of lateral ventricle; F_3, inferior part of third frontal convolution; *l. stric*, lenticulo-striate division of internal capsule; *Knie. ic*, knee of internal capsule; *l. optic*, lenticulo-optic division of internal capsule; *Th*, optic thalamus; *J*, island of Reil; *cl*, claustrum; *Operc*, operculum; T_1, first temporal convolution; *r. lic*, retrolenticular region of internal capsule; *C. A.*, ammon's horn; *calc*, calcarine fissure; *Hh*, posterior horn of lateral ventricle; *SS*, optic radiation of Gratiolet; T_2, second temporal convolution; *Facialis*, position in capsule of motor tract to the face; *Hypoglossus*, position of tract to the tongue; *Arm*, position of tract to the arm; *Bein*, position of tract to the leg; *S. B.*, sensory fibres; *S*, visual tract; *A*, auditory tract.

oculomotor palsy with or without hemianopsia is characteristic of a lesion of the crus. It has been termed Weber's syndrome. The eye affected is, of course, the one on the side of the lesion, and hence on the side opposite to the paralysis of the face, arm, and leg. It is turned outward, cannot be moved upward, downward, or inward, and its pupil is dilated. Ptosis is present, that is, the upper lid falls, and the eye is closed by paralysis of the levator palpebræ.

Hemiplegia may be due to a lesion in the upper part of the pons, but when the lower part of the pons is affected the face escapes on

FIG. 174.



Scheme of the motor tract to show the effect of the lesions at different positions. 1. Cortical or subcortical lesion, causing monoplegia of left arm. 2. Capsular lesion, causing left hemiplegia. 3. Crus lesion, causing left hemiplegia and right third nerve palsy. 4. Pons lesion, causing alternating paralysis of right face and left arm and leg. S. Sylvian fissure. O.T. Optic thalamus. N.L. Lenticular nucleus. C. Crus. N.C. Caudate nucleus. VII. Facial nerve. M. Medulla.

the side on which the limbs are paralyzed. This is because of the decussation of the tract to the facial nucleus which enters the raphe in the pons and crosses to the other side. But such a lesion interrupts the course of the tract to the facial nucleus on the side of the lesion. Hence from lower pons lesions we get *alternating paralysis;* that is, a paralysis of the face on the side of the lesion and of the arm and leg on the opposite side. If the tract to the facial nucleus is affected the eye can be closed. If the nucleus itself is destroyed the eye cannot be closed. This form of paralysis cannot be caused by a lesion elsewhere

The motor tract to the hypoglossal nucleus which governs the tongue pursues a course analogous to that of the facial nucleus, but crosses the middle line at the upper part of the medulla. A condition of paralysis of one-half of the tongue and of the arm and leg on the opposite side is produced by a lesion in the medulla; such lesions are rare.

The decussation of the motor tract takes place in the pyramids of the medulla. It is incomplete, some fibres crossing to the lateral tract of the cord, others going down in the anterior median column of the same side. The degree of the decussation varies in different persons. In some it is almost total, in others it is partial. In some it is irregular, many fibres crossing on one side and few on the other. Thus Fig. 35 shows a marked asymmetry in the lateral tracts and anterior median columns due to such irregular decussation. Flechsig has remarked this difference in fœtal cords, and has even found one case in sixty in which no decussation occurs at all. This variability in the anatomy of the motor tract explains the different degree of paralysis in different cases of hemiplegia. In some cases the paralysis is very complete on one side and hardly perceptible on the other. In other cases there is distinct weakness on the side opposite to the hemiplegia. A few rare cases have been reported by Brown-Séquard in which the hemiplegia was on the side of the lesion, and in these we may assume that there was no decussation of the motor tract. A lesion of the medulla may affect both tracts at their decussation, and hence cause paralysis of all four limbs. This, again, is very rare.

The motor tract in the spinal cord in the lateral column may be affected in the disease lateral sclerosis which has been already studied. A unilateral affection of the tract in the spinal cord is not common, hence a hemiplegia from spinal lesion is extremely rare.

These types of hemiplegia occur from every kind of brain disease. The alternating form is indicative of a lesion on the base either within or outside of the brain axis.

Disturbance in the sense of touch is a local symptom of disease in the brain. There is some discussion as to the area of the cortex which receives these sensations and as to the course of the tract which brings them in.

The probable localization of the sensory centres is in the posterior central convolution and in the adjacent portions of the cortex of the parietal lobules. Lesions in this locality usually cause a loss of tactile sense in the opposite side of the body. The sensory area can be divided, like the motor area, into subdivisions for face, arm, and leg, which lie respectively in the lower, middle, and upper thirds of this region. A collection of cases of local cortical disease made by me in 1884 led to this conclusion. Dana subsequently collected other cases which supported it, and Nothnagel, Charcot, Gallet, and von Monakow accept this localization.

Some authorities have held that the sensory and motor areas exactly coincide. Dejerine, Flechsig, Henschen, Mott, and Schäfer urge this view. But my cases prove that lesions in the motor area in front of the fissure of Rolando rarely cause loss of tactile sense, while lesions behind it usually produce anæsthesia.

Another theory of the localization of sensory centres, held by Ferrier and Mills, places them in the hippocampal region. This has been founded on physiological experiment, but is not confirmed by pathological records in man, and those who hold it have not attempted any subdivision of this area for the different limbs. I think it may be discarded, for in many cases of operation upon the cortex in living persons for the relief of epilepsy and for the removal of tumors I have seen small lesions of the cortex of the convexity near the motor area cause a loss of sensation, sometimes temporary, sometimes permanent.

This loss of sensation is usually limited to one side of the face or to one limb in cases of cortical disease for the same reason that paralysis in cortical disease is more commonly of the monoplegic than of the hemiplegic variety. A loss of sensation in the entire side of the body, termed hemianæsthesia, is more commonly caused by a lesion of the sensory tract within the brain than from disease of the cortex.

Irritation of the cortical centres for touch causes a sensation of tingling in the part of the body which corresponds to the centre affected. This is termed paræsthesia. It has been already alluded to as a frequent warning and as a constant accompaniment of Jacksonian epilepsy, under which circumstances it is a temporary symptom. It is frequently observed as the result of slight disturbances of circulation in the cortex, and hence is a prodrome of apoplexy. It occurs as a permanent symptom in cases of pressure on the cortex, and hence is found in cases of cortical clot and of cortical tumor. The tingling, when temporary, is often followed by a slight temporary anæsthesia, and when permanent is always attended by a slight tactile anæsthesia.

Disturbance of the sense of touch includes both superficial sensations of touch, temperature, and pain, and deep sensations of articular and muscular sense. There is no question that these different elements in the sense of touch are independent of one another, and it is certain that they are transmitted to the cortex by different sensory tracts which we shall presently trace. There is every reason to believe that they are received in different neurones of the cortex, for clinical experience teaches that one sense may be lost when others are preserved. Thus in one of my cases of apoplexy the patient lost the sense of temperature alone on one side; in another case the patient could perceive cold sensations only, and had a constant feeling of cold, but could not perceive warm sensations; in still another case sensations of temperature and pain were lost, while touch was preserved in one side. In two cases[1] of operation upon the cortex I

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[1] One of these is reported in the American Journal of the Medical Sciences, Nov., 1894, with McCosh. The second case has been recently seen with him at the Presbyterian Hospital.

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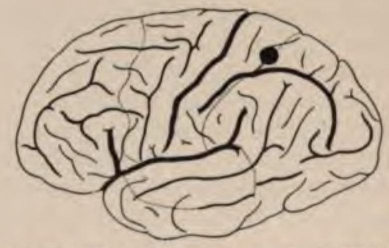
FIG. 175.



Situation of a tumor causing loss of muscular sense and ataxia in the right hand, with astereognosis.

sense are independent of one another, so far as our present knowledge goes, it is necessary to locate them grossly in the same locality, viz., near to, but behind, the motor zone. It is possible that various layers of the cortical neurones possess different functions, but this is only a hypothesis.

The characteristics of cortical hemianæsthesia are its common association with hemiplegia, the limb which is most paralyzed being most anæsthetic. But there is no necessary relation between motor and sensory disturbance, as either may occur alone, or one may be intense while the other is slight.

The anæsthesia from cortical lesion is never total. Each side of the body is connected with both hemispheres of the brain, though the crossed connection is far more complete than the direct one. Hence a slight degree of sensibility in the anæsthetic side is always to be found, especially in cases of any length of duration. Cortical anæsthesia usually subsides gradually after a lesion, and the sensibility returns in part even if not wholly. Dejerine noticed that this was more common in young persons than in middle-aged or old ones. The anæsthesia from organic lesions is always most intense in the distal part of a limb; the fingers and hand are more insensitive than the forearm, and this than the arm or trunk; the toes and sole are more insensitive than the leg, and this than the thigh; the face is more insensitive than the neck or trunk, and the lips are the most insensitive part of the face when it is affected. There is no sharp limit to the anæsthesia, it fades gradually into an area of moderate sensation. Dejerine calls attention to all these characteristics as enabling one to distinguish it from the anæsthesia occurring in hysteria. This latter is often found without paralysis, is absolute, does not disappear gradually, is of uniform intensity in the entire side, and is often associated with concentric diminution in the visual field of the eye on the anæsthetic side.

Tests of Sensation. Touch is to be tested by cotton-wool or by a camel's-hair brush or by the fingers of the examiner, the two sides of the body being touched simultaneously in symmetrical places, and the patient being requested to compare the two sensations. Temperature sensations may be similarly tested, test tubes with hot and cold water being used. Pain sense may be tested by needles or by a faradic brush. Muscular sense may be tested by the use of differently weighted balls or coins held in the hands and compared, or by weights being placed on the hands which are supported, each side being tested separately or the two together. The sense of position may be tested by giving a position to one limb or hand and asking the patient to reproduce this in the other side. If, for example, the right hand is put in a clenched position and the blindfolded patient cannot detect that he must clench the left one in order to imitate this position he has a loss of muscular sense in the right hand, for the error lies not in the power of movement but in the perception of the kind of motion necessary. A further test may be made by demanding the execution of accurate and co-ordinated movements, such as picking up a pin, buttoning the clothes, carrying a glass of water to the mouth.

The cortical sensory area, in the act of handling any object, receives simultaneously numerous tactile and temperature sensations associated with the sensations of pressure and of weight and of the positions of the fingers involved in feeling the object. These sensations leave behind them a permanent trace in some physical change in the cortical cells. They are combined into a sensory memory of the object, so that following the acquisition of this complex sensory mental picture we can recognize the object by the sense of touch, even if we do not see it. Thus we have tactile memories of objects such as an orange, a rose, a bell, a pen, which enable us to call them to mind and to recognize them when felt. This power of recognizing objects by touch has been named stereognosis. It is evident that the stereognostic sense is not a simple thing, but involves a simultaneous activity in a large number of different tactile sensory neurones which are grouped together. The recognition of an object by it is comparable rather to the recognition of a melody in music as distinguished from a single note. This is one of the functions of the portion of the cortex concerned in tactile sensations. And in fact, when we consider our sensations for a moment we perceive that it is always some definite object which we recognize in perceiving a tactile sensation rather than the sensation alone. It is the touch of a finger, of a pin, of cotton-wool, of a hard weighty object rather than touch or firmness or weight which we feel. These abstract qualities of objects are only known by a process of analysis and synthesis, a process of generalizing from a large array of particulars. The thing which we primarily feel and recognize by touch is not a quality, it is an object. It is necessary, therefore, to regard the sensory area of the cortex as the storehouse of tactile memories of objects just as we regard the motor area as the storehouse of motor memories of combinations of motion. The loss of the power of recognition by touch has been named astereognosis.

The majority of our tactile perceptions are acquired through the hand. We have few memories of facial or pedal sensations, and practically none of the trunk. Hence the tactile memory area is more extensive in the middle third of the posterior central convolution and in the adjacent part of the inferior parietal lobule. (Fig. 176.) Redlich[1] (1893) collected twenty cases of lesions limited to this area which had been attended by astereognosis, and Walton[2] has recently supported this view by other cases. The area involved is somewhat higher than the supramarginal gyrus, which obviates the objection of Dejerine that in lesions there he has not seen the symptom.

A loss of tactile memories may therefore occur as a symptom of disease in the sensory area of the cortex, and it is a valuable localizing symptom. It forms a part of the condition of apraxia, to which we shall allude later. It is the essence of the condition of astereognosis.

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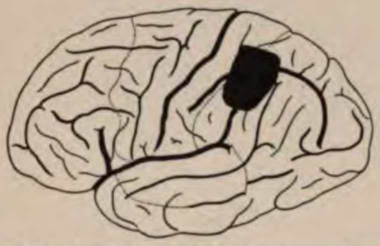
[1] Wiener klin. Wochen., 1893.

[2] Brain, 1901.

Astereognosis is to be elicited in a patient by placing various objects one after another in the hand, the eyes being closed, and asking their names and uses. Each hand must be tested separately as each hemisphere contains its own set of memories. If aphasia is present names may not be given, but by signs a patient can usually indicate whether he recognizes an object and its uses by touch.

FIG. 176.



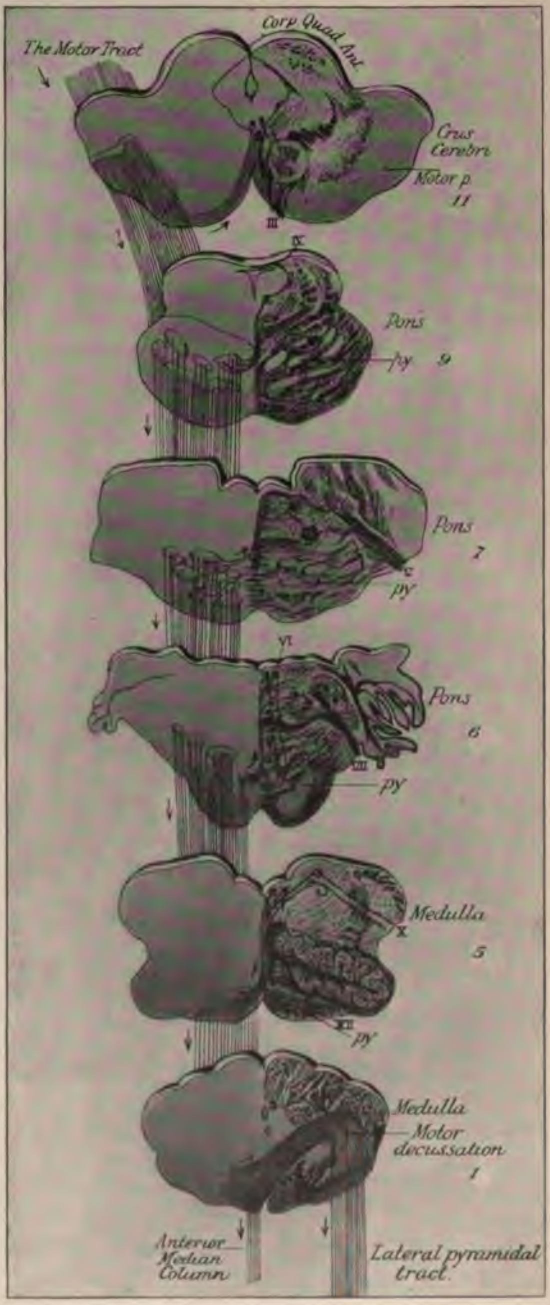
The area of the cortex in which a lesion causes astereognosis.

Sensory Symptoms from Subcortical Lesions. Hemianæsthesia, hemianalgesia, hemithermo-anæsthesia, and hemiataxia may be produced not only by lesions in the sensory area of the cortex, but also by an interruption in the sensory tracts conveying these sensations from the body.

In studying diseases of the spinal cord we have already seen that these sensations ascend by separate tracts. Tactile sensations ascend partly in the posterior columns and partly in the antero-lateral tracts. Temperature and pain sensations ascend in the antero-lateral ascending tract. Muscular sensations ascend partly in the posterior columns and partly in the direct cerebellar columns. (See Figs. 42 and 43, page 186.)

It is necessary to trace these various tracts upward from the cord to the cortex of the sensory area. The sensory tracts are not as direct and continuous as the motor tracts through the nervous system. The study of ascending degenerations in the spinal cord, cerebral axis, and brain has shown that such degenerations, though intense and extensive just above a transverse lesion, become less so the higher we get, and hence we conclude that the short tracts greatly outnumber the long ones. In fact, we do not find continuous sensory tracts from the cord to the cortex. We have a series of short connecting tracts interrupted by gray masses, and a moment's consideration shows the reason of this anatomical arrangement. The effect of a sensation in any part of the body, especially if it be of sufficient importance to give rise to pain, is not merely a conscious perception. The primary effect is a multitude of reflex actions entirely below the sphere of consciousness. Thus an ordinary painful impression results

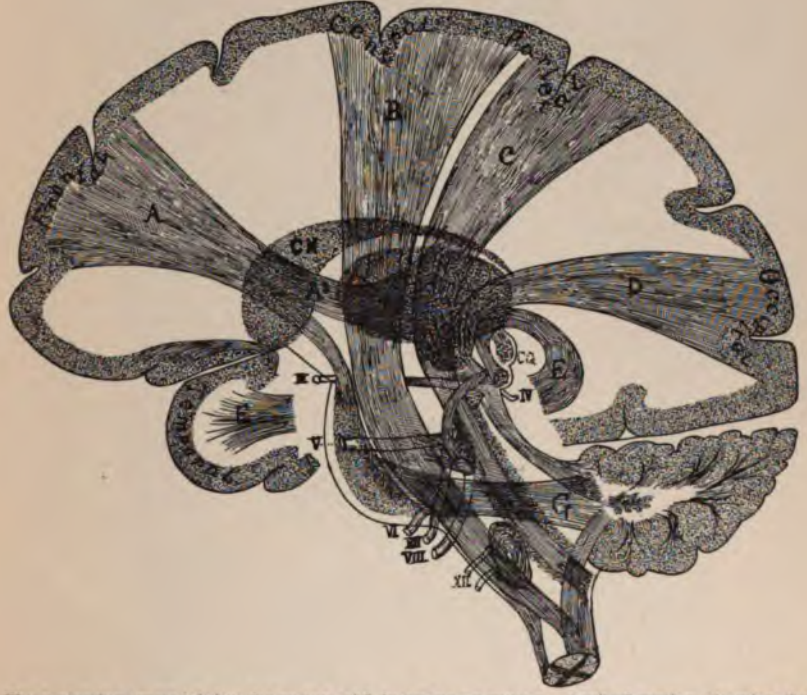
FIG. 177.



The cerebral axis. The course of the motor tract (py).

in the withdrawal of the limb, in setting up of vasomotor and trophic reflex acts, or, if the irritation be kept up, of a general sense of uneasiness throughout the body, even complex reflex acts to remove the source of pain, a quickening of respiration and pulse, a facial expression of discomfort, and even automatic cries. All these effects can be produced in an animal whose cortex is removed and in a man asleep or under ether. It is therefore evident that an intense sensory impression may throw into activity the entire subcortical nervous system, or, in other words, may be distributed to a multitude of motor mechanisms in cord and cerebral axis and subcortical ganglia in addition to awakening cortical activity and causing a conscious perception. This effect could not be attained were the sensory tracts from one region of the body to the cortex continuous and single. It could only be attained by an arrangement which provides for the

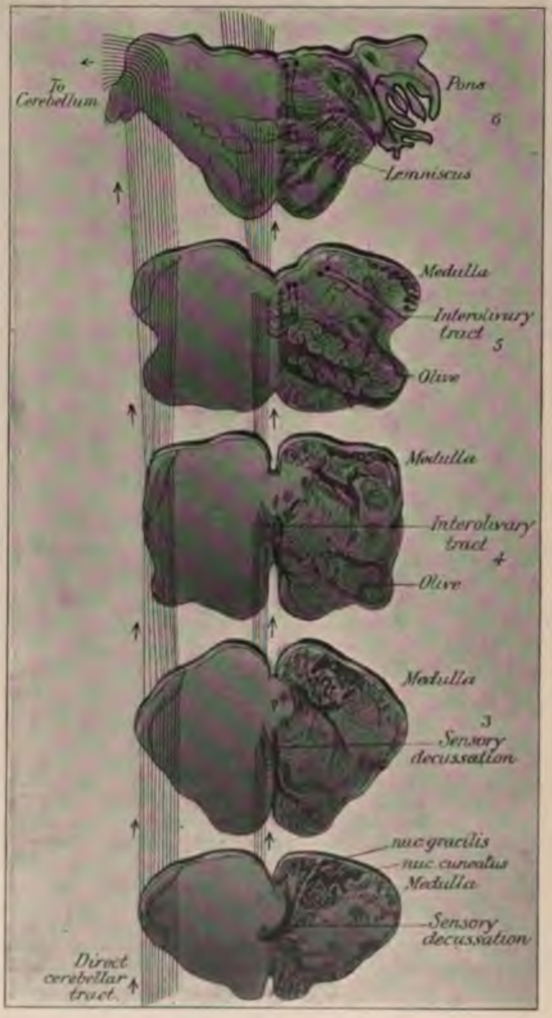
FIG. 178.



The projection tracts joining the cortex with lower nerve centres. Sagittal section, showing the arrangements of tracts in the internal capsule. A, tract from the frontal lobe to the anterior half of the capsule, thence in part to the optic thalamus, A², and in part to the pons, and thus to the cerebellar hemisphere of the opposite side; B, motor tract from the central convolutions to the facial nucleus in the pons and to the spinal cord; C, sensory tract from posterior columns of the cord, through the posterior part of the medulla, pons, crus, and capsule to the parietal lobe; D, visual tract from the optic thalamus (OT) to the occipital lobe; E, auditory tract from the int. geniculate body (to which a tract passes from the VIII. N. nucleus) to the temporal lobe; F, superior cerebellar peduncle; G, middle cerebellar peduncle; H, inferior cerebellar peduncle; CN, caudate nucleus; CQ, corpora quadrigemina. The numerals refer to the cranial nerves.

termination of a single sensory tract at various levels, a mechanism which disseminates a single sensation to a number of different motor centres. The diagram given on page 26 (Plate II.) shows the existence of a sensory tract made up of a number of segments each joined to the next, but each having interpolated between it and the next a neurone capable of turning aside some of the impulses received into side channels.

FIG. 179.

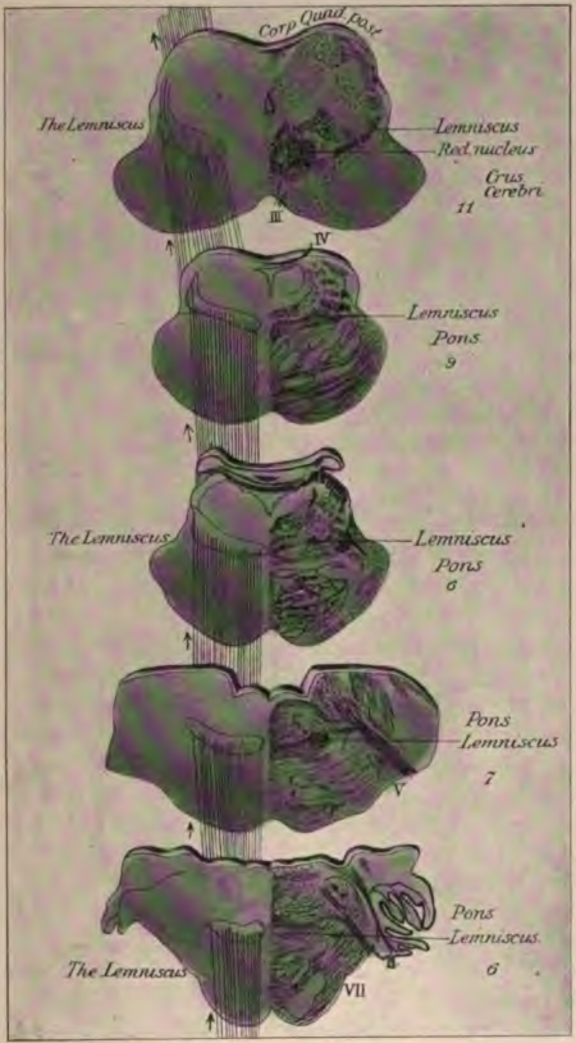


The tract of muscular sense—the interolivary tract.

The Sensory Tract. With these facts in mind it is possible to trace the sensory tract upward and to understand the symptoms which will present themselves when it is injured. It will then be clear that from the distribution of the symptoms of anæsthesia it is often possible to locate a lesion in the sensory tract.

1. **The Tract of Muscular Sense.** The posterior columns of the cord end in the nuclei gracilis and cuneatus of the medulla (Fig. 179), each axone terminating in a brush about the neurone bodies which form these nuclei. From these neurones axones pass upward, crossing over in the sensory decussation to form the opposite interolivary

FIG. 180.



The tract of muscular sense—the lemniscus.

tract which lies between the olives. The hypoglossal nerves on their way from the nucleus to their exit lie on the outer side of this tract. Ascending in this tract the sensory fibres enter the lemniscus, which lies in the pons Varolii behind the pyramidal tracts and deep transverse fibres. Many axones branch from the lemniscus to end about neurones lying in the gray matter of the pons, both in the floor of the ventricle, in the formatio reticularis, and in the deep gray matter. From these neurones again axones enter the lemniscus to ascend. The lemniscus ascends (Fig. 180) through the crus cerebri, where it curves about its lateral surface, sending many of its axones into the corpora quadrigemina posterior and anterior, then sinks into the base of the brain, and, passing through the lower part of the internal capsule, terminates largely in the optic thalamus in its outer nucleus. (Fig. 178.) It is possible that all the lemniscus fibres end there. It is possible that some pass up through the capsule and turn outward to end in the cortex of the posterior central convolution and adjacent parietal region. It is possible that the only sensory fibres entering the cortex come from the optic thalamus, but many authors have traced the lemniscus directly to the cortex, at least in part.

In 1884 I showed,[1] by an analysis of twenty-six cases of very small local lesions limited to the medulla and pons, that an interruption in these fibres either (1) in the posterior columns of the cord; (2) in the interolivary tract; (3) in the lemniscus, or (4) in the internal capsule where the lemniscus passes produces a condition of ataxia and loss of the muscular sense in the limbs of the opposite side. Since that date many similar cases of small lesions in the lemniscus have been reported with the same result. Hence it may be stated that this is the tract of muscular sense and that a lesion of this tract causes ataxia.

If, therefore, hemiataxia, not of cortical origin, occurs, the conclusion can be drawn that the lemniscus is affected. The position of the lesion in the course of the lemniscus can be determined only by a study of other symptoms coincident with the ataxia. As the medulla, pons, and crura are very small and contain the nuclei of the various cranial nerves at different levels some symptoms referable to an affection of one of these cranial nerves will usually enable one to locate the lesion. Thus, if there is oculomotor paralysis with hemiataxia the lesion is in the crus. If there is trigeminal anæsthesia or facial palsy with hemiataxia the lesion is in the pons. If there is auditory or hypoglossal paralysis the lesion is in the medulla. The figures indicate the course of this tract and its relation to the various cranial nerves.

2. **The Tract of Tactile, Pain and Temperature Senses.** The ascending sensory fibres in the lateral columns of the spinal cord which convey these sensations pass directly into the corresponding portion of the medulla oblongata which is known as the formatio reticularis.

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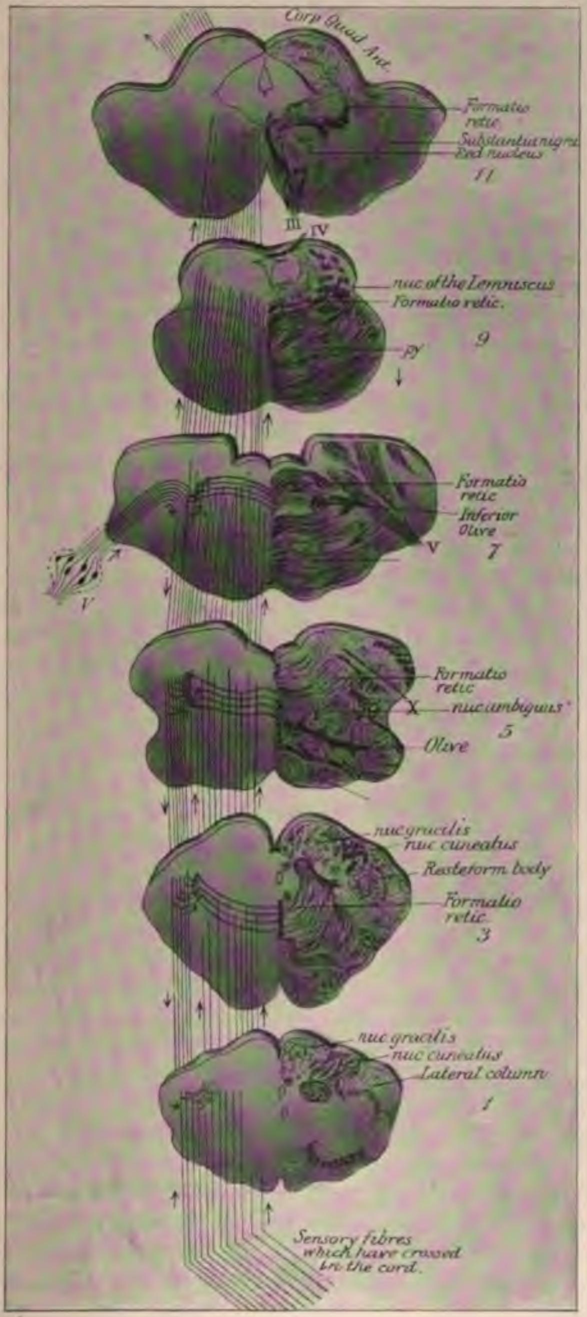
[1] Journal of Nervous and Mental Disease, July, 1884.

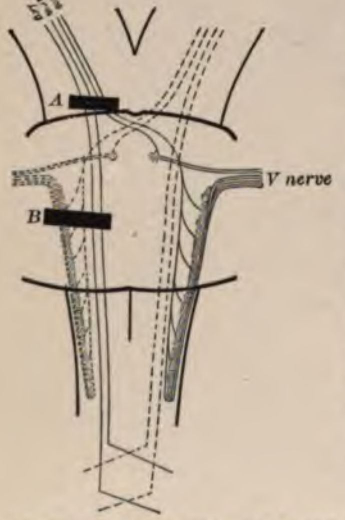
Fig. 181.



The sensory tract through the cerebral axis.

This is shown in Fig. 181. It is made up of the interlacing of three sets of fibres: (*a*) transverse, the commissural fibres of the medulla, pons, and cerebellum; (*b*) vertical, the cranial nerve fibres; (*c*) longitudinal, the sensory tract; and hence, however it is cut, it appears as a network or reticular formation, whence the name. It is strewn with isolated neurone bodies and has collections of these at many points; thus the nucleus ambiguus, the superior olive, the nuclei of the lemniscus, and the red nucleus are collections of neurones lying in this formation. The formatio reticularis is continuous from the medulla through the pons and into the crus, whence its fibres enter the internal capsule. Through this formatio reticularis many fibres pass up and some fibres end. From it many ascend through the crus to enter the optic thalamus. The formatio reticularis, therefore, contains a long ascending tract. The sensations sent along this from the spinal cord, like those in the lemniscus, pass either indirectly by way of the thalamus or directly by way of the internal capsule to the sensory cortex. The ascending tract of Gowers in the spinal cord takes the same course into the formatio reticularis, but its termination is not yet certain.

FIG. 182.



The sensory tract in the crus, pons, and medulla, showing nucleus and roots of V nerve. *A*, lesion causes right hemianæsthesia; *B*, lesion causes alternating hemianæsthesia; left face and right side of body.

In the outer third of the formatio reticularis is found a column of small cells of peculiar structure, resembling the substantia gelatinosa of the posterior horn of the spinal cord, and in this column terminate the fibres of the sensory part of the trigeminal nerve (Fig. 182) which turn downward after entering the pons Varolii. Thus the outer portion of the formatio reticularis contains the sensory tract from the face of the same side.

Lesions of the formatio reticularis interrupt the sensory tract and cause anæsthesia. It is evident from the diagram that a lesion which involves the formatio reticularis on one side of the pons and medulla will produce an alternating anæsthesia—*i. e.*, loss of sensation in the face on the side of the lesion and in the body on the other side. Alternating anæsthesia is as characteristic a symptom of lesions of the formatio reticularis of the pons and medulla as alternating paralysis is of lesions in the motor tract in the pons.

In the upper part of the pons the sensory tract from the face crosses the median line, and hence a lesion in the formatio reticularis in the upper third of the pons, or in the crus cerebri, or in the internal capsule will produce a unilateral hemianæsthesia. A lesion involving both halves of the formatio reticularis will produce bilateral sensory symptoms.

The diagrams show the relation of this tract to the cranial nerves which make their exit through the formatio reticularis. A lesion which is situated in the crus, pons, or medulla, causing anæsthesia of the body, will also cause some symptoms referable to one or more of the cranial nerves, and hence such a lesion can readily be localized.

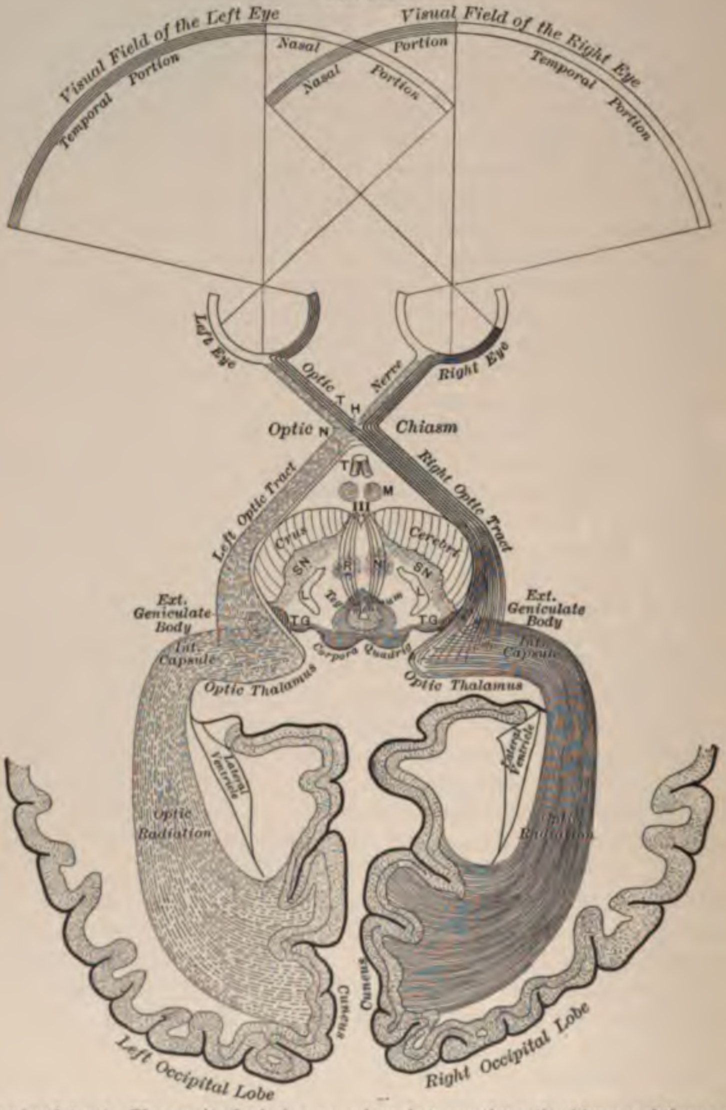
Hemianæsthesia is usually due to a lesion of the sensory tract in the internal capsule, where it lies either just behind the motor tract or mingled with it. Such a hemianæsthesia is usually attended by hemianalgesia and hemithermo-anæsthesia and often by hemiataxia. A dissociation of sensations, that is, the loss of either touch, pain, or temperature sense alone, the others being intact, has not been observed after lesions of the internal capsule. Hemianæsthesia from lesions of the capsule is rarely absolute, for the sensory decussation is not a complete one, and sensations from one-half of the body are sent to both hemispheres of the brain. As a rule, the entire half of the body is anæsthetic after a lesion of the capsule. But as the sensory tract ascends to the cortex from the capsule its fibres spread out like the sticks of a fan, those transmitting sensations from the leg passing upward to the summit of the sensory area of the cortex, while those transmitting sensations from the face pass outward to the vicinity of the Sylvian fissure. Hence in lesions in the centrum ovale, as in lesions of the cortex, one limb or part of the body may be anæsthetic instead of the entire side. And the same rule applies to symptoms of loss of sensation as to paralysis: that the nearer the lesion to the cortex the more likely are the symptoms to be limited to a single limb. Thus in the cases shown in Figs. 167 and 168 there was a loss of sensation as well as of motion in the hand and arm of the opposite side.

When the close approximation of the sensory to the motor tract throughout its course is considered it is no longer a matter of surprise that in the majority of cases of brain disease a loss of sensation attends paralysis. It is to be noticed, however, that in all this course the sensory tract lies behind the motor tract. If, therefore, a lesion is advancing from behind forward in the brain sensory disturbance precedes paralysis, but if the lesion is advancing from before backward, paralysis precedes anæsthesia. This observation has an important bearing upon diagnosis, for it sometimes enables one to determine whether the disease under view is a progressive or a stationary one, and, if progressive, to settle the direction in which it is extending. (See Fig. 178.)

3. **The Cerebellar Tract from the Spinal Cord.** The direct cerebellar tract from the cord turns through the corpus restiforme of the medulla outward and goes directly to the cerebellum. (Fig. 179.) Some fibres from the nuclei gracilis and cuneatus of each side join it in the corpus restiforme and end with it in the cerebellum. It is also joined

by fibres from the opposite olivary body. These tracts, therefore, do not reach the sensory cortical centres, and hence impulses passing along them have nothing to do with sensations of touch, temperature, and pain, or with those muscular sensations which are destined to

FIG. 183.



The visual tract. The result of a lesion anywhere between the optic chiasm and the cuneus is to produce homonymous hemianopsia. H, lesion at chiasm causing bilateral temporal hemianopsia; N, lesion at chiasm causing unilateral nasal hemianopsia; T, lesion at chiasm causing unilateral temporal hemianopsia; SN, substantia nigra of crus; L, lemniscus in crus; RN, red nucleus; III, third nerves.

guide voluntary movements. They are concerned in the transmission of those muscular sensations which are essential to the control of equilibrium, as we shall see later when we consider the connections and functions of the cerebellum. It is not to be forgotten, however, that in lesions affecting the medulla and pons this tract may be injured, and thus vertigo and staggering may be produced.

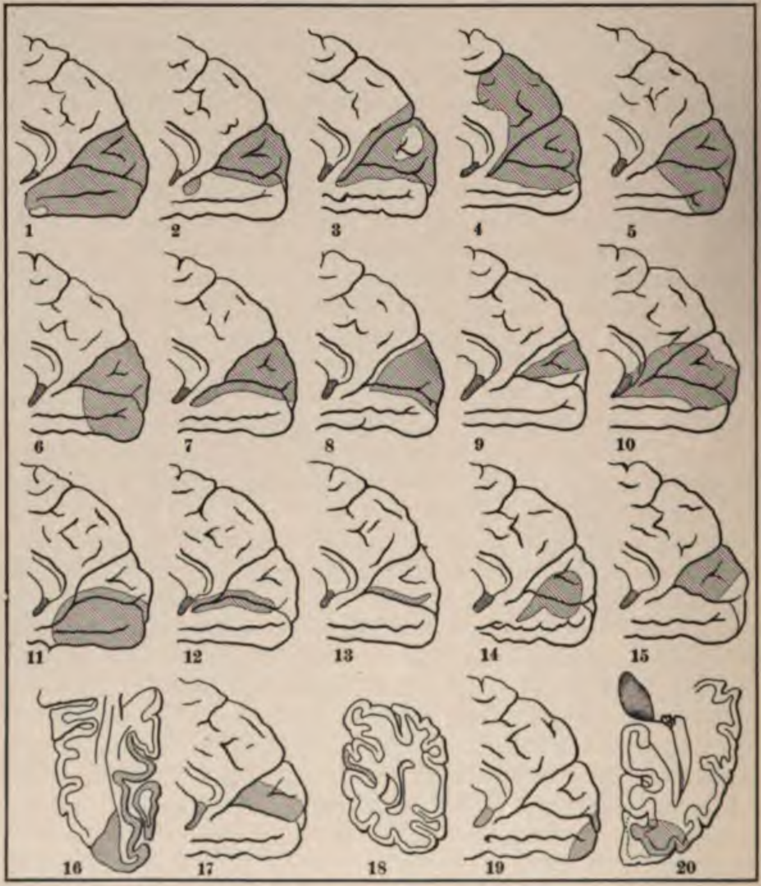
Disturbance of vision is a local symptom of cortical disease of the occipital lobe of the brain and of the visual tract from the eyes to it. The optic nerves pass from the eyes to the optic chiasm. A partial decussation of the optic fibres occurs in the optic chiasm, leading to the junction in the optic tract of fibres from the corresponding half of both eyes. For this reason cerebral blindness is always a hemianopsia or blindness in one-half of both eyes, each hemisphere of the brain receiving impressions from the opposite visual fields. The exact anatomy of the optic nerves and optic tracts will be considered in Chapter XXXIV. Suffice it here to mention that each optic tract ends in the pulvinar of the optic thalamus, in the external geniculate body, and in the corpus quadrigeminum anterior (Fig. 183), and that from these ganglia the visual tract issues into the posterior fifth of the internal capsule, and turning backward in it passes through the centrum ovale, outside of the posterior horn of the lateral ventricle, and terminates in the cortex of the occipital lobe. The exact termination of these fibres is in the cortex about the calcarine fissure and in the cuneus, a wedge-shaped lobule on the median surface of the hemisphere. (Fig. 184.) But the convolutions of the convexity of the occipital lobe are also concerned in the reception and storing up of visual impressions, and hence lesions in any part of the occipital cortex are productive of hemianopsia.

There seems to be a projection of the visual field to some extent upon the cortex of the cuneus, for small lesions there may cause small sector-like defects in the visual field. Henschen, in his elaborate work on the pathology of the brain, has confirmed this conclusion, which Wilbrandt and Hun had reached from a smaller number of cases. Fig. 184, *17*, shows the location of the lesion in Hun's case. The defect in the visual fields in this patient was limited to the upper quadrant. Lesions in the upper part of the cuneus cause blindness in the lower quadrant of both visual fields, and lesions in the lower part of the cuneus cause blindness in the upper quadrant of both visual fields. It is rare for a lesion, however, to be so exactly limited as to cause this effect. As a rule, lesions anywhere in the occipital lobe produce hemianopsia. Superficial lesions in the occipital cortex may cause a loss of color vision only, hemichromatopsia, which may be partial, that is, limited to one or two colors only, or total. Such cases are very rare and are usually soon followed by hemianopsia. A few cases have been reported of bilateral lesions causing double hemianopsia. In one such case central vision was preserved (Forster), in others the blindness was total. Total blindness has also been caused by a maldevelopment of the occipital lobes of the brain, as in cases reported by Haab and Spiller.

Lesions of the cortex produce defects of the visual field that are symmetrical in both eyes, as shown in Fig. 185.

Psychical Blindness. The loss of the power of vision is attended by a loss of visual memories when the lesion is cortical, and hence the recognition of objects seen is imperfect, and the mind cannot recall the appearance of things formerly familiar. Thus I have known patients who, after such a lesion, did not recognize the members of their family by sight, though they recognized them by the sound of their voices. This condition is known as psychical blindness. The patient fails to recognize objects, and cannot recall them to mind.

FIG. 184.

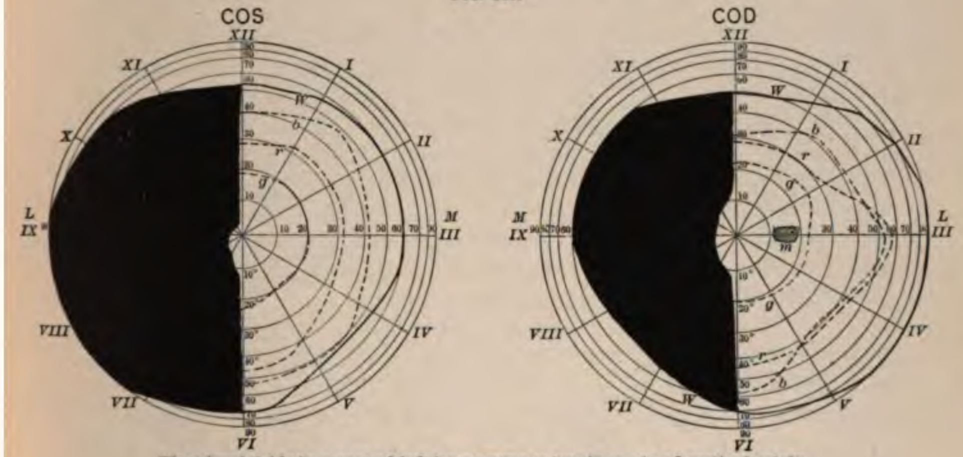


Lesions of the occipital lobe causing hemianopsia. (From Henschen.)

It would seem, from a study of more or less extensive lesions in the occipital cortex as if the memories of things seen were largely located in the left hemisphere in right-handed and in the right hemisphere in left-handed persons, thus according with the location of our memories of speech. At any rate, the loss of these memories is much more frequent when the left hemisphere is destroyed.

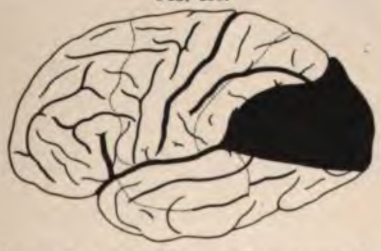
The major part of our knowledge of objects is acquired through the sense of sight, and a moment's thought will convince one not only that the extent of cortex occupied by memory pictures must be great, but also that it must be increasing daily through life. The only definite conception we can have of this process is that, while all objects are originally perceived through impressions sent to the cuneus, the memories of these objects are stored in secondary centres outside of the cuneus in the occipital cortex. Hence the larger the number of things seen and remembered the greater the area concerned in vision in any individual.

Fig. 185.



The visual fields in a case of left homonymous hemianopsia of cortical origin.

Fig. 186.

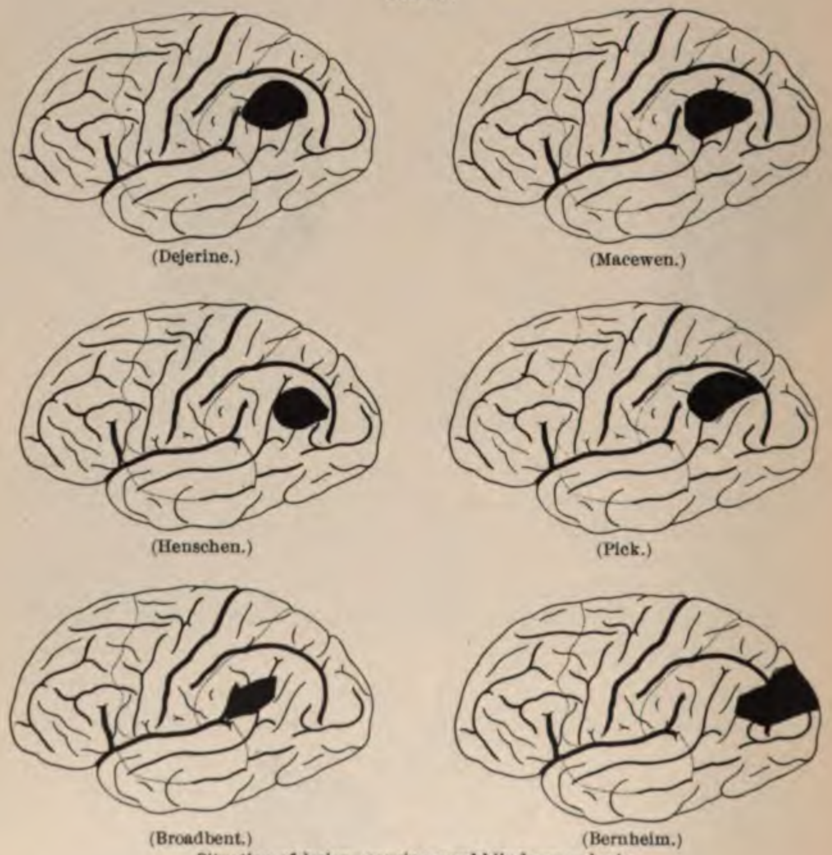


The area of the cortex in which a lesion causes psychical blindness.

The condition known as psychical blindness is due to a loss of these memories and is diagnostic of a lesion of the cortex of the occipital lobe. It occurs from a lesion of the convexity as well as from a lesion of the cuneus. It is usually attended by hemianopsia, always when the cuneus is destroyed. (Fig. 186.)

Word-blindness. There is a special class of visual memories, those that make up our knowledge of written and printed language, which have a special location. This is in the junction of the occipital and parietal regions in an area known as the angular gyrus. Lesions in this locality cause a loss of the memory of words as seen; hence an inability to read. This symptom, while really a part of psychical blindness, is termed word-blindness, and may occur without psychical blindness. It is often accompanied by hemianopsia, because a lesion in this locality, if not altogether superficial, may involve the visual tract which lies beneath the cortex here. This is shown in Fig. 183. But word-blindness may occur alone without hemianopsia. It may be attended by hemianæsthesia or hemiataxia, but in such cases the lesion involves the cortex in front of the angular gyrus. Fig. 187 shows the exact extent of the lesion causing word-blindness in six cases. Patients suffering from word-blindness not only lose the power of reading, a condition termed alexia, but sometimes lose also

FIG. 187.


(Dejerine.) (Macewen.)

(Henschen.) (Pick.)

(Broadbent.) (Bernheim.)

Situation of lesions causing word-blindness; alexia.

the power of writing, a condition termed agraphia, being unable to call up the proper motor images needed to form the word. Yet there are cases on record where alexia has occurred without agraphia, and I have seen two patients who could not read what they had just written. In such cases one must imagine that the sound of the word as mentally pronounced awakened the motor memory of its production in speech and in writing, even when its appearance could not be recalled. Word-blindness is an important division of sensory aphasia.

Visual Aura. Irritation of the occipital cortex produces forced revivals in consciousness of visual memories. These may consist simply of flashes of light or color or of actual images. They are known as visual hallucinations when consciousness accepts them as real and does not distinguish them from actual objects seen. Such hallucinations often precede a Jacksonian epilepsy and are known as visual auræ. They are, as a rule, unilateral, the hallucination appearing on the side opposite to the lesion. They are analogous to the tingling sensations known as the signal symptom in localized spasm. They are sometimes followed by temporary hemianopsia, the excitement of the cortical neurones causing an exhaustion.

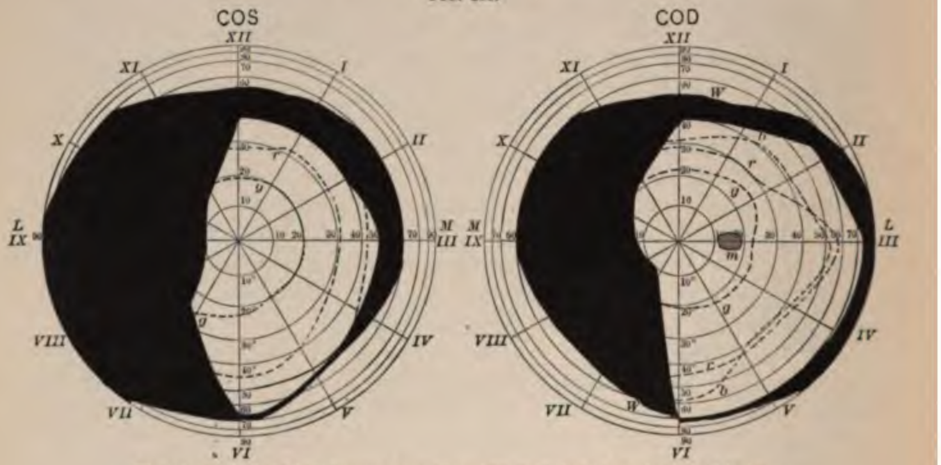
It is probable that the visions of delirium, whether due to poisons, such as alcohol, opium, or cannabis indica, or due to inflammation of the membranes of the brain or of the brain itself, are produced by irritation of the visual area of the cortex.

Subcortical Hemianopsia. Hemianopsia may be due to a lesion in the visual tract beneath the cortex or to a lesion in the basal ganglia in which the optic tract terminates. In this case the limit of blindness in the visual field is usually less extensive and less symmetrical in the two eyes than when the lesion is cortical. In cortical hemianopsia the line of limitation of vision is usually vertical and passes within one or two degrees of the point of central vision in both eyes. In subcortical hemianopsia the line is irregular and passes from five to ten degrees outside the point of central vision in both eyes. (Fig. 188.)

Hemianopsia due to subcortical lesions is usually attended by other symptoms. Thus if it is right hemianopsia and the lesion is just beneath the cortex at any point in the temporo-occipital radiation some form of word-blindness or aphasia is a very common symptom. If the lesion is in the region of the internal capsule or optic thalamus, hemiataxia or hemianæsthesia is very often an associated symptom. This is due to the proximity in this locality of the sensory tract. Hemiplegia may also accompany it from the same cause. If the hemianopsia is due to a lesion in the corpus geniculatum externum the fibres of the corpora quadrigemina are involved and the reflex acts of the pupils are affected as they are in lesions of the optic tract. Then the hemiopic pupillary reflex of Wernicke can be elicited. (See Chapter XXXIV.) Lesions limited to the corpora quadrigemina, while causing disturbances in the movement of the two eyes, double vision, and imperfect pupillary reflexes, do not, when alone, cause any loss of vision. They produce vertigo and a staggering gait, like cerebellar disease, from implication of the red nuclei lying under the corpora quadrigemina.

Hemianopsia may be due to a lesion in the optic tract on one side. It is then associated with Wernicke's hemiopic pupillary reflex. (See Chapter XXXIV.) The optic tract lies upon the crus, and lesions affecting it often simultaneously affect the motor tract, and cause hemiplegia, and also involve the oculomotor nerve, causing ptosis and double vision (see page 432). The forms of hemianopsia due to lesions in the optic chiasm are described in Chapter XXXIV.

FIG. 188.



The visual fields in left homonymous hemianopsia due to a subcortical lesion.

Lesions of the occipital lobe, or of the internal capsule, or of the basal ganglia do not cause blindness of one eye, as was formerly held by Charcot and Ferrier. A blindness in one eye or a concentric diminution of the visual field of one eye is due either to functional disturbance (hysteria) or to a lesion of the optic nerve, visible by the ophthalmoscope.

Disturbance of hearing is rarely due to disease in the brain unless this disease affects both temporal lobes or the tracts to them. Each ear is connected with both temporal lobes, hence a unilateral lesion fails to cause total deafness, and there is no such thing as a partial cortical deafness to high or low sounds or tones which might be analogous to hemianopsia or hemichromatopsia. A cortical deafness, therefore, has not been observed excepting in a few rare cases where both temporal lobes have been destroyed. In these cases the patient has become totally deaf. The exact localization of the auditory centres is in the middle part of the first temporal convolution, and extends to the adjacent cortex within the Sylvian fissure and over the convexity into the second temporal convolution. In cases of congenital deaf mutes these parts are found to be atrophied. In some cases of irritating lesions of this area Jacksonian attacks of epilepsy have been preceded by an auditory aura, loud sounds, bells, whistles, etc., being heard just as the attack was coming on.

Psychical Deafness. Our auditory perceptions, like those of sight, leave behind them a trace which constitutes our auditory memory. We have such memories of sounds, of musical melodies and harmonies, and also of words heard. Hence lesions in the auditory area may deprive a patient of these memories, even though he may retain his power of hearing. It is an extraordinary fact that our memories of things heard appear to be stored in one hemisphere only, the left in right-handed and the right in left-handed persons. Hence psychical deafness and amusia and word-deafness are symptoms of a unilateral lesion of the temporal cortex.

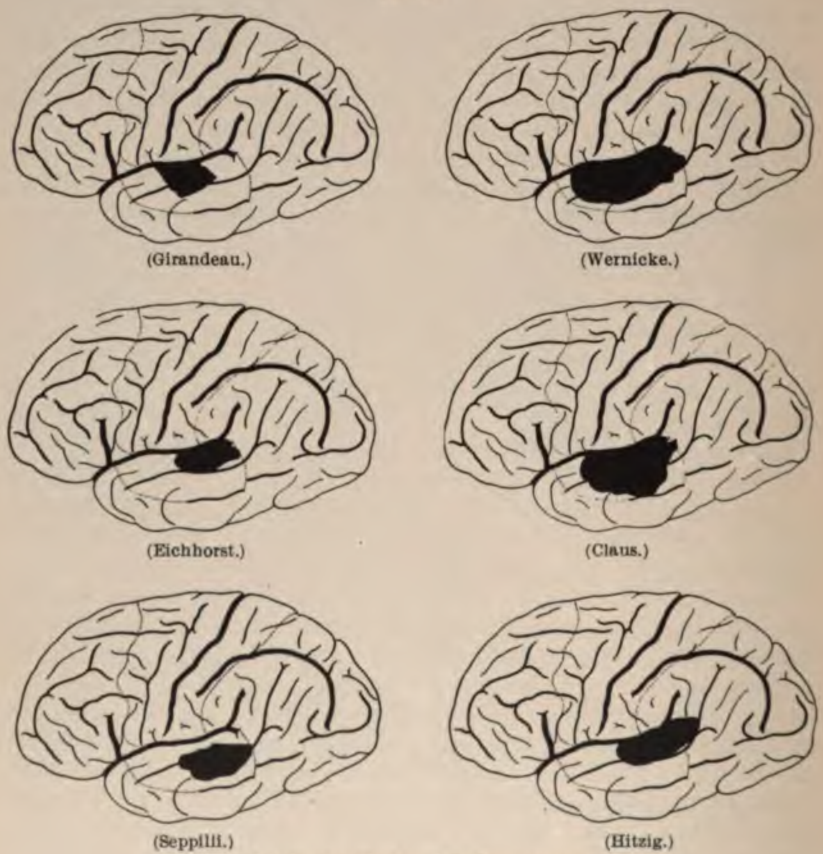
A patient with psychical deafness no longer recognizes any noise or sound. The bark of a dog, the cry of any animal, the sound of waves, or bells, or whistles, the voice of a familiar person no longer awakens recognition, and such sounds cannot be called to mind.

A patient with amusia, which is a variety of psychical deafness, no longer recognizes melodies and harmonies with which he was familiar. He no longer takes pleasure in hearing music or operas in which he may formerly have delighted, and all music seems to him new and strange. He cannot call to mind familiar tunes, and hence cannot sing or hum them.

Word-deafness. A patient with word-deafness, which may occur independently of or in connection with amusia and mind-deafness, is no longer able to recall the names of objects, and is no longer able to understand spoken language. He is like one who hears an unknown tongue. He hears the sounds of voices, but attaches no meaning to what is said. He may be able to read and he may be able to speak in the sense of saying words correctly, but he usually talks jargon, as the sounds no longer guide his talking. This is the second and more common form of sensory aphasia.

Our auditory memories of words are the first to be acquired in infancy, and long before an infant can talk he has learned to associate the sound of names with their corresponding objects. The memory pictures of objects, acquired chiefly by sight, but also by all other senses, are the fundamental basis of thought in all animals, and these, grouped together, form the concept of each object which they know. To this concept is soon joined by association the sound of its name; later, in man, the motions needed to produce this sound are learned; lastly, the appearance of the letters making this word, and finally, the motions necessary to write the word. The auditory memories are not only the first speech elements acquired, they are the most important. Reading and writing are subordinate to speech, a result of the later evolution of language, and are an evidence of education, not of natural acquisition. The sound of objects in nature has been taken to indicate these objects in the early evolution of speech, and is still so in the education of many infants. The baby learns "bow-wow" before it learns "dog." The study of language reveals that a large number of words—*e. g.*, murmur, whisper, puff, bang—have a similar origin.[1] As our knowledge of the names of objects is constantly growing, as we acquire new languages, it is evident that our auditory memories are constantly increasing in number. We must believe, therefore, that the cortical area in which they are stored varies in size in different persons, according to the degree of education in language. This is proven by cases of gradually advancing diseases in this area. I have a patient who, as such disease went on, lost first his memory of English, then of German, and finally of French, the latter being his native tongue. The names of objects first acquired appear to persist longest in memory. Thus a patient

FIG. 189.



(Girandeau.) (Wernicke.)

(Eichhorst.) (Claus.)

(Seppilli.) (Hitzig.)

Situation of lesions causing word-deafness; sensory aphasia.

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[1] French. On the Study of Words. Macmillan & Co.

who is word-deaf will often comprehend single words when he cannot understand a sentence, and one finds one's self unconsciously talking to an aphasic very much as one talks to an infant. The condition of word-deafness may be and usually is incomplete, some words being recognized and remembered and others forgotten. Some patients retain nouns, but forget adjectives and verbs and prepositions. Others lose all nouns and talk in roundabout phrases. Thus one of my patients could not name a knife or a scissors, but said "it is the thing you cut with." Occasionally all the word-memories are lost and nothing that is heard is understood.

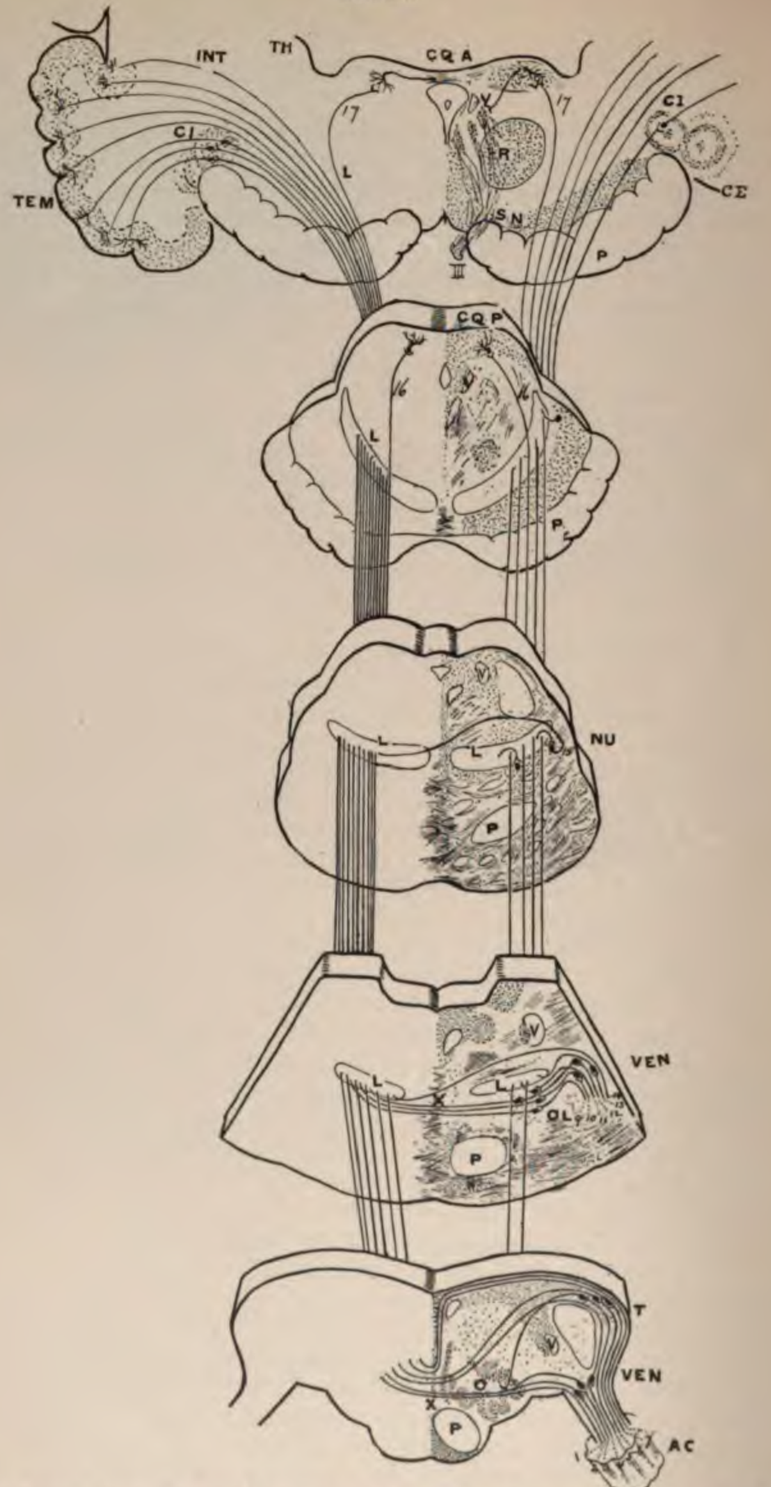
The lesion of word-deafness is located in the cortex and subcortical region just beneath it in the first and second temporal covolutions, an area a little wider than that concerned in hearing alone. Fig. 189 shows the location of the lesion in six cases of this kind.

The Auditory Tract. Deafness from subcortical lesions is a rare symptom. Fig. 190 shows the course of the auditory nerve impulses from their entrance into the medulla to their termination in the cortex.

The neurone bodies, of which these fibres are the axones, lie in the spiral canal of the cochlea. It is the cochlear division only of the eighth nerve which has to do with hearing.

AC, the acoustic nerve fibres, enter the side of the medulla in a large trunk. We can distinguish fourteen different sets of fibres. Many of these fibres (1–8) penetrate the medulla opposite the entrance of the nerve. Others (9–14) turn upward on entering, and penetrate the pons at a little higher level. These various fibres pass to different destinations; 1 crosses through the olive, turns upward in the inter-olivary tract, and enters the opposite lemniscus, and passes up in it; 2 terminates about a neurone body in the ventral nucleus (VEN). From this body an axone arises which crosses to the opposite side and turns upward in the lemniscus; 3 terminates about a neurone body in the ventral nucleus (VEN). From this body an axone arises which passes upward in the lemniscus of the same side; 4 terminates about a neurone body in the ventral nucleus (VEN). From this body an axone arises which passes about the outer side of the medulla through the tuberculum acusticum (T), and thence through the formatio reticularis of the medulla to the raphé, where it crosses to the opposite side and turns upward in the lemniscus; 5 terminates about a neurone body in the dorsal nucleus or tuberculum acusticum (T). From this body an axone passes through the formatio reticularis into the lemniscus of the same side and turns upward in it; 6 terminates about a neurone body in the dorsal nucleus (T). From this body an axone passes through the formatio reticularis, crosses in the raphé and enters the lemniscus of the opposite side, turning upward in it; 7 terminates about a neurone body in the dorsal nucleus (T). From this body an axone passes across the floor of the fourth ventricle in the striæ acusticæ to the raphé, turns downward in it, crosses to the other side, enters the lemniscus and turns upward in it; 8 passes

FIG. 190.



The auditory tract. TH, optic thalamus; INT, int. capsule; CI, corp. geniculatum int.; TEM, temporal lobe; L, lemniscus; CQA, corp. quadrigeminum ant.; CQP, corp. quad. post.; R, red nucleus of tegmentum; SN, substantia nigra; CE, corp. geniculatum ext.; P, pes pedunculi and pyramidal tract; NU, nucleus of lemniscus; VEN, ventral nucleus of auditory nerve; V, fifth nerve; OL, olivary nucleus; O, olive; X, decussation of auditory fibres; AC, auditory nerve from *cochlea ; T, dorsal* nucleus of auditory nerve.

through the dorsal nucleus into the striæ acusticæ and accompanies 7 in its course.

The ventral nucleus of the auditory nerve consists of a long column of cells which extend upward into the pons. Hence in a section through the lower half of the pons the ventral nucleus is still visible (VEN) and the fibres ascending from the auditory nerve trunk to terminate in or to pass through this nucleus (fibres 9–14) are easily traced. The course and ending of these fibres is as follows:

No. 9 turns inward and ends about a neurone body lying in the upper olivary nucleus (OL). From this body an axone arises which crosses the median line in the trapezium, enters and ascends in the opposite lemniscus; 10 terminates about the neurone body in the ventral nucleus, whence a new axone arises and passes to the olivary nucleus, terminating about a neurone body there. From this neurone body an axone arises which joins 9 and pursues the same course; 11 passes through the ventral nucleus and ends about a neurone body in the mass of gray matter lying adjacent to but ventral of the lemniscus, and dorsal of the olivary nucleus. This is the trapezoid nucleus. From its neurone bodies axones arise, some of which enter the lemniscus of the same side, but many of which cross the median line to enter the lemniscus of the opposite side and ascend in it; 12 terminates in the ventral nucleus about a neurone body. This body sends its axone to the collection of neurones lying within the deep transverse fibres of the pons, the trapezoid body. These neurone bodies in turn send their axones into the lemniscus of the same and of the opposite side; 13 terminates about a neurone body in the ventral nucleus. The body sends an axone directly into the lemniscus of the same side; 14 passes through the ventral nucleus and crosses in the trapezoid from the opposite side, where it turns upward in the lemniscus.

It is thus evident that all the fibres of the acoustic nerve, so far as its cochlear division is concerned, transmit their impulses into the lemniscus of the same or of the opposite side. The trapezoid fibres may be termed the acoustic decussation or chiasm, and, as in the optic chiasm, the majority of the fibres cross to the opposite side (X). The termination of fibres ascending in the lemniscus is very complex. (*a*) Some fibres terminate about the cells of the nucleus lemnisci in the pons (Nu) which nucleus in turn sends axones to the corpora quadrigemina of the same and of the opposite side (15). (*b*) Some fibres terminate about the large quadripolar cells of the posterior corpus quadrigeminum (16) (CQP). (*c*) Some fibres terminate about the large cells of the first layer of cells in the anterior corpus quadrigeminum (17) (CQA). (*d*) Many fibres terminate about neurone bodies in the corpus geniculatum internum (CI), whence new axones arise which pass to the cortex of the temporal lobe (TEM). (*e*) Some fibres pass directly through the internal capsule from the lemniscus to the temporal lobe.

Since each of the nuclei in which lemniscus fibres terminate is connected with motor mechanisms as well as with the cortex of the temporal lobe, it is evident that the auditory impulses can awaken numerous reflex and automatic acts as well as conscious sensations of hearing. Hence the act of turning eyes and head or assuming postures of strained listening and other automatic acts are made possible by these fibres.

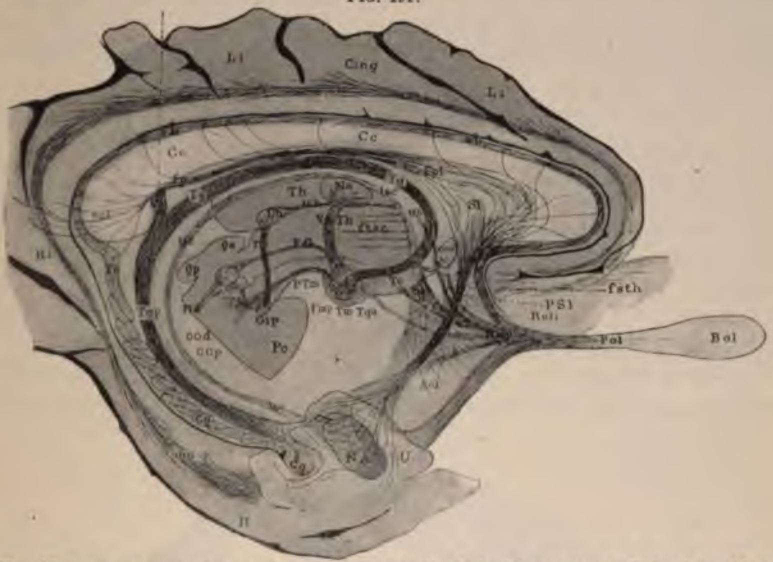
Fig. 190 shows that the connection of each ear is with both sides of the brain, but that the crossed connection is more extensive than that with the same side. The diagram does not show the existence of a corresponding set of neurones whose axones pass in the direction the reverse of those shown. Degenerative changes after experimental injuries prove their existence. Hence a second diagram might be drawn showing axones of exactly complementary course. These are omitted from this diagram for the sake of clearness.

It is evident that a lesion limited to one lemniscus will not cause deafness any more than a lesion limited to one temporal lobe. There are cases on record, however, in which the lemniscus on both sides has been affected in pons lesions and in these deafness has occurred. In six out of twenty-four cases of limited lesions in the pons, which I collected some time ago, there was deafness. Hence in any case where a pons lesion is suspected from its characteristic symptoms, viz., alternating paralysis (see page 433), alternating anæsthesia (see page 446), or cranial nerve palsies (see Chapter XXXIV.), it is well to test for deafness, and if it is found to regard it as a local symptom of disease of the lateral part of the lemniscus in the cerebral axis.

Disturbance of the sense of smell is occasionally elicited in diseases of the brain. This sense, which is of vital importance to animals, being their chief guide to food, is of little importance to man, and hence its organs are gradually undergoing retrograde evolution. In the mammals, and especially in carnivora, the olfactory bulb and its cerebral tracts are enormous and are the best protected of all parts of the brain from injury, lying deep within the cranium. Fig. 191, from Dejerine, shows the numerous connections of the olfactory bulb and fibres and their relations to the subcortical ganglia, and their termination in the uncinate gyrus and nucleus amygdalus which lie at the apex of the temporal lobe. It is in this area that physiologists have located the sense of smell in the cortex. It is not improbable that here, too, is located the sense of taste, its tract coming from the optic thalamus by way of the trigonum. The taste impulses can reach the thalamus from the fifth nerve nucleus by way of the formatio reticularis. (See Chapter XXXIV.)

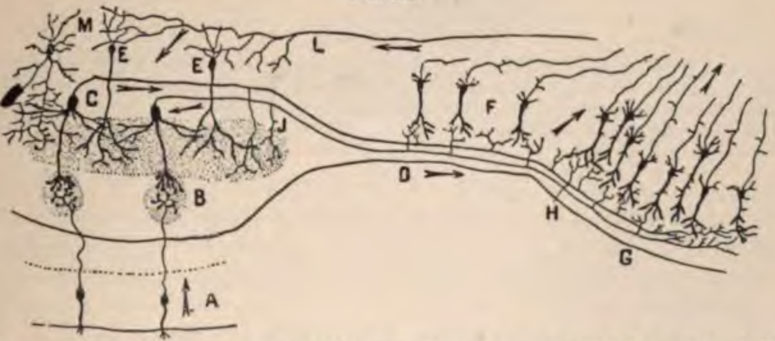
There are a few cases on record in which irritation of the apex of the temporal lobe has caused hallucinations of smell or of taste. Such hallucinations may precede Jacksonian epileptic attacks, as in a case of Hamilton and one of Jackson, in both of which local meningitis had affected both uncinate gyri. Destructive lesions of this area with loss of smell have not as yet been recorded. The usual cause of a loss of smell, when not due to nasal disease, is some local lesion on the base in the frontal lobe, destroying the olfactory bulb or tract.

FIG. 191.



The olfactory system and tracts. Bol, olfactory bulb; Pol, olfactory tract; Rolp, deep olfactory fibres; Roli, internal olfactory fibres; Role, external olfactory fibres. The deep fibres pass to the thalamus (Th) and its nuclei; Tc, tuber cinereum; Tm, tuber mammillare, and to tsc, tænia semicircularis, and tth, tænia thalami; also into the anterior commissure, coa. The internal olfactory fibres pass into the striæ of Lancisi, sL, and thus to the fasciola cinerea, Fc. The external olfactory fibres pass directly to the uncinate gyrus, U. Tg, fornix from the corpus mammillare (Tm) to Ammon's horn (Cg); NA, nucleus amygdalæ; Cing, cingulum; Cc, corpus callosum; Fc, fasc. cinerea; FG, Gudden's fasciculus; FM, fasc. retroflexus of Meynert; Na, ant. nuc. of thalamus; Gip, interpeduncular ganglion; Gb, ganglion habenula; Qa, Qp, corp. quadrigemina; Po, pons; VA. fascic. Vicq. d'Azyr. (Dejerine.)

FIG. 192.



The olfactory bulb and tract. A, Schneiderian membrane in nose in which lies peripheral olfactory neurone; B, glomerulus of olfactory bulb; C, mitral cells with dendrites in B and axones in D olfactory lobe; E, granule cells; F, cells in olfactory lobe; H, G, fibres of olfactory tract. (Ramon y Cajal.)

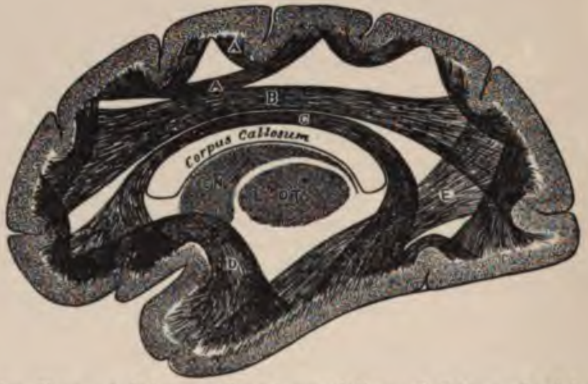
Disturbance of the sense of taste as a local symptom of brain disease has not been observed. The nerves concerned in this sense are the fifth and ninth, and a loss of taste must still be referred to their affection. (See Chapter XXXIV.)

We have memories of smell and taste which are undoubtedly stored in the cortex of the uncinate gyrus and which go to make up the concepts of objects which possess odor and flavor. We have no record of cases of psychical anosmia, though such symptoms are perfectly possible.

Disturbance of Thought and in the Use of Language. Thus far the symptoms produced by a lesion in one functional area of the cortex or in the tract leading to it, or from it through the brain have been considered, and also the possible combinations of these symptoms when adjacent areas or tracts are involved together. But while the cortical areas have undoubtedly distinct functions, it is not to be forgotten that they are closely joined to one another by means of the multitude of association fibres and commissural fibres which go to make up the larger part of the white matter of the centrum ovale.

It can be shown by careful dissection that each convolution is joined to the two adjacent convolutions by fibres which pass around the separating fissures. (Fig. 193.) Also, that bundles of fibres exist which pass from each convolution to the convolution next but one, and so on. Hence, it may be stated that each convolution has a possible connection with every other. Besides this association of convolutions by small bundles of fibres, it is possible to find a distinct set of association tracts which pass between more or less distant regions. One such tract passes from the frontal lobe, collecting its bundles from all three convolutions, backward to the occipital lobe. Another tract joins the occipital with the anterior part of the temporal lobe. Another passes from the upper two temporal convolutions forward to the third frontal convolution, passing

FIG. 193.



The association fibres. A, between adjacent convolutions; B, between frontal and occipital areas; C, between frontal and temporal areas, cingulum; D, between frontal and temporal areas, fasciculus uncinatus; E, between occipital and temporal areas, fasciculus longitudinalis inferior; CN, caudate nucleus; OT, optic thalamus.

beneath the island of Reil. And a tract from the frontal to the posterior temporal area may also be found. Their relative degree of development in animals is directly in the ratio of the cortical development, and their actual number is an index of mental capacity.

The function of these association fibres is to form the physical basis for the association of concrete memories and of psychical acts, and their integrity is necessary to thought. By studying subjectively the association of ideas in the mind the importance of their function becomes evident.

We have already seen that each sensory impression leaves a memory picture which is the basis of our recognition of an object. But each object produces on the mind a number of different impressions simultaneously. These are, therefore, associated together in the mind and form a *concept* consisting of numerous memory pictures joined together by means of the association fibres in the brain. These fibres form as essential a part of concepts as the various memory pictures. Thus a single memory picture has little meaning unless it is associated with others; a new object seen has to be brought into relation with objects already known before it becomes understood or fixed in the mind. It must also be brought into relation with its name. And thus the simplest object, "a rose" or "a bell," is

FIG. 194.

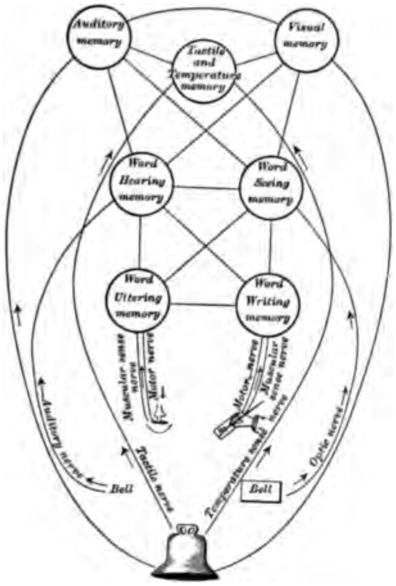


Diagram to illustrate the concept "bell" and to show the varieties of apraxia and aphasia. The memory pictures are relics of past perceptions received through different senses. Their association makes up the mental image bell. The word image is made up of the memories of the sound and appearance of the word, and of the uttering and writing-effort memories; these are joined together. The mental image and the word image are also joined with one another, making up the concept "bell." (Charcot.)

known only when the numerous sensory impressions which it awakens are united into a complex mental substratum. We have no single ideational centre or concept centre in the brain, but each concept is made up of many pictures. This is shown in the diagram of Charcot of the concept bell. (Fig. 194.) When we hear a peal and it awakens the memory of the word "bell" and also the appearance of a large church bell or small dinner bell, whose shape and cold, hard, metallic feel and weight can be called to mind, and when we recollect the corresponding word in French or German or any other language that we know, and also recall the effort needed to pronounce the corresponding word in our own and in the foreign tongue, and also to write it and to read the letters that make up the word, we have merely aroused in consciousness one after another a number of dormant memory pictures by sending impulses over association fibres that unite these pictures together. Hence, association fibres are necessary to thought, and a break in them or a destruction of their terminal stations will impair mental action.

The loss of concepts has been termed *apraxia*. The loss of speech has been termed *aphasia*. We have already considered the varieties of apraxia, viz., psychical blindness, psychical deafness, amnesia, astereognosis.

Aphasia. The symptom *aphasia* requires further consideration. We have already seen that the basis of language is a series of memory pictures; (1) of the sound of a word; (2) of the effort necessary to enunciate it; (3) of the appearance that its printed or written symbols present; and (4) of the effort needed to produce these symbols in writing. These memory pictures are intimately connected with each other by means of association fibres which pass in all directions between the areas in which they are located. They are also joined to the memory pictures which make up the concept. They are so intimately joined that if one is lost all the others feel the effect, and a break in the mechanism leads to a defect in the act of speech.

The defects of speech are broadly divided into sensory and motor aphasia, according to the predominant feature of the defect. Sensory aphasia consists in a lack of power to recognize or to recall the sound or appearance of words. If the sound alone is lost the condition is termed word-deafness; if the appearance is lost the condition is termed word-blindness. Motor aphasia consists in a lack of power to initiate the effort and set in action the motor mechanism needed to pronounce or to write a word. The former is termed motor or ataxic aphasia, the latter motor agraphia. They are usually associated.

1. WORD-DEAFNESS. If the memory of the sound of the word is lost the word cannot be called to mind and cannot be recognized when heard. Show the patient a watch, and he is unable to name it; tell him it is a stone, a match, a watch, and notice whether he dissents from the former and gives signs of satisfaction at the last. If he does he has only auditory amnesia or partial word-deafness. He

retains the sound memory picture, and it can be aroused by hearing it; hence the lesion is not in the cortex where the memory lies, but he cannot make the connection between the sight of the object and it. Here the memory picture of the sound of its name can be awakened directly through the ear—that is, by one of the tracts to the cortex, if not by another. Hence in such a case the lesion must be subcortical. If, however, he does not recognize the sound of the name when heard he is totally word-deaf and is unable to understand what is said to him. Such a patient, therefore, is in the condition of a man who hears a foreign language; he hears sounds, but attaches no meaning to them. He makes no associations between the word and its concept. He does not understand. This condition usually very much impairs his use of words, for he is unable to call to mind the sound of the word, and therefore is unable to say it. But the motor act of pronunciation may be initiated if the impulse can be sent to its centre without passing through the auditory centre. This is the case in exclamations, when one does not choose his words; hence many patients can swear, though they cannot talk. If, in a patient with word-deafness, there is no accompanying word-blindness, he may be able to read aloud as well as to himself—that is, his motor speech memory may be aroused by way of his visual memories without the intervention of the auditory memories. And if he has no apraxia it is also possible for any of the concept memories to awaken the motor speech memory; hence the thought of an object or seeing it may lead to the enunciation of its name without thought of how the name sounds For this reason patients who are word-deaf and cannot understand what is said to them may be able to talk fairly well.

The lesion in total word-deafness is located in the middle part of the cortex of the first and second temporal convolutions in the left hemisphere in right-handed, and in the right in left-handed persons. (See Fig. 189, page 456.)

2. WORD-BLINDNESS. If the memory of the appearance of the word is lost, the visual image of it cannot be called to mind or recognized, and then the patient will be unable to read, because the shapes of the letters and words seen arouse no recollection. This is also termed alexia. He will also be unable to write spontaneously, for he cannot remember how the letter that he wishes to write looks. As a matter of fact, words are forgotten more easily than letters, and if a patient is to relearn to read he must begin with letters, and go on to words. Figures are sometimes recalled when words are forgotten, and many a patient can do mathematical calculations on paper who cannot read or write ordinary words. The reverse may also be true, the patient being able to read and write, but being unable to understand or to write figures or to calculate. Such patients may play cards or other games, if they are not psychically blind. It is not infrequently the case that persons who are thus word-blind can write at dictation, or copy, and yet show no evidence of understand-

ing what has just been written. Here the writing centre has been called into activity through some association tract without the intervention of the word memory picture. A distinction must be made between those who have lost the memory picture and those in whom it cannot be recalled by ordinary means. The first have cortical word-blindness; the second have subcortical word-blindness. The word memory picture may be cut off from its ordinary channels of connection with other memory pictures and yet remain intact. In such a case some roundabout road to it will lead to its revival in consciousness, and it will be found to be preserved, though inaccessible by ordinary means.

The condition of visual amnesia with word-blindness is due to a lesion involving the inferior parietal convolutions and angular gyrus, and is often associated with psychical blindness, but may occur independently of it. (See Fig. 187, page 452.)

Word-deafness and word-blindness frequently occur together, and then the lesion is found involving both the temporal convolutions and the angular gyrus.

3. OPTICAL APHASIA OR INTERCORTICAL SENSORY APHASIA. When the association-fibres between the memories of sight and the memories of sound are severed a condition of aphasia results which is characterized by an inability to recall the name of a thing seen and to picture to the mind the appearance of a thing named. Yet the name is recognized when heard and the object is recognized when seen. This condition has been described under different names by different observers. Freund[1] named it optical aphasia or transcortical aphasia, and these terms are used by the Germans.[2] I prefer the term intercortical sensory aphasia as less obscure and misleading. A patient suffering from this type of aphasia has not lost his memory pictures, for he is able to recognize anything once heard or seen. He can, therefore, hear, understand, and read; but if he is asked to call to his mind some place or person whose name is given—*e. g.*, Lake George, Lake Como, President McKinley—he cannot do so. The impulse started from the word-hearing centre cannot reach and arouse the visual memories; nor can the association be made in the opposite direction, for if he is shown an object or a person—a watch, a chain, or some familiar face—he cannot recall the name, though he recognizes it when heard.

There are numerous cases on record with autopsies which prove that the lesion in this condition lies in the long association tract within the temporal and occipital lobes. This tract is shown in Plate XIX. Such a lesion may be an abscess of the brain secondary to ear disease, or a tumor, or a focus of softening. In a case described in the chapter upon abscess of the brain this symptom was made the basis of a surgical operation which proved successful. Should an extensive cortical lesion occur in the convolutions between the temporal and

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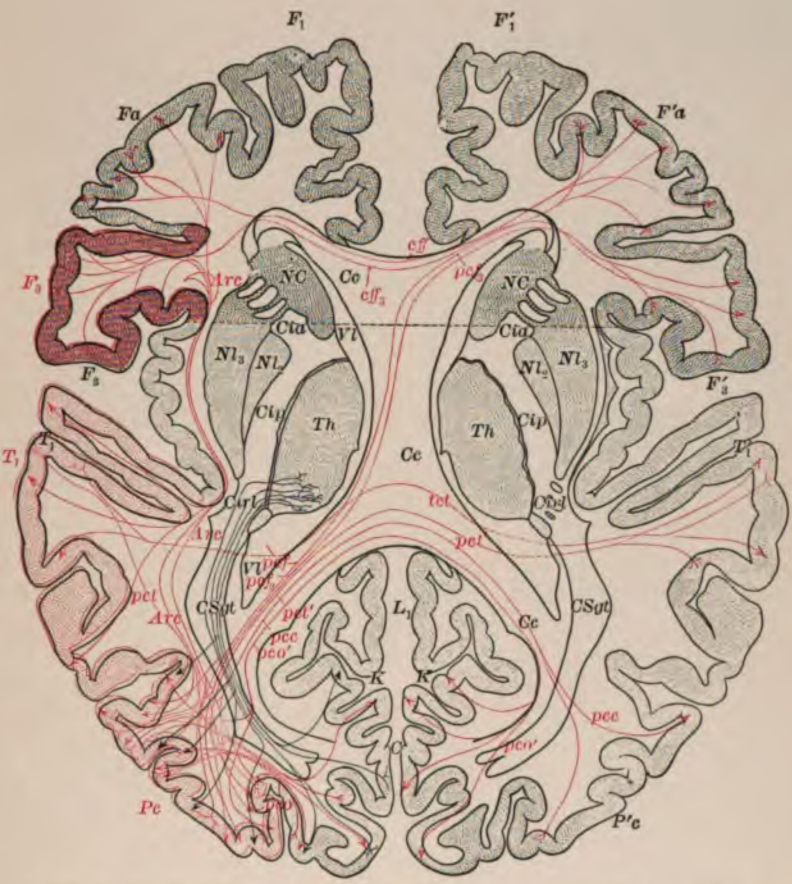
[1] Freund. Arch. f. Psych., xx., 276.

[2] Vorster. Arch. f. Psych., xxx., 341.

PLATE XIX.



The Connections of the Areas of the Cortex Included in the Zone of Language. (Dejerine.)

The upper part of the figure represents a vertical section through the brain. The lower part represents a horizontal section below the transverse dotted lines. *F*, frontal; *Fa*, anterior central; F_3, Broca's; *T*, temporal; *Pc*, angular convolutions; ***Arc***, arcuate fibres joining the angular gyrus *Pc* and the temporal convolutions T_1 to Broca's convolution F_3 and to the motor centre for the arm *Fa*; *pct*, association fibres between the angular gyrus and temporal convolutions; *pcc*, association fibres between the two hemispheres; *pcf*, fibres joining the left angular gyrus to the right motor centre; pcf_3, fibres joining the left angular gyrus to the right third frontal convolution; *pco*, *pco'*, fibres joining the left angular gyrus to the occipital convolutions of the left and right lobes; *tct*, commissural fibres between the two temporal lobes; *Th*, thalamus; *NC*, caudate nucleus; *Nl*, lenticular nucleus; *Cia*, internal capsule, anterior limb; *Cip*, posterior limb; *Cirl*, *CSgt*, visual tract; *Vl*, lateral ventricle; *cff*, commissural fibres in corpus callosum *Cc*; *C*, cuneus.

occipital gyri and invade the white matter beneath them the same symptoms would be produced.

4. Motor Aphasia. If the memory of the effort needed to pronounce a word is lost, a true paralysis of active speech occurs, though the muscles may not be weakened. This is the ordinary form of motor aphasia, due to a lesion of Broca's centre in the posterior part of the third frontal convolution on the left side in right-handed persons. It is to be noted that such a loss of speech involves a loss of the power of repeating words after another, as well as of voluntary speech, and is not accompanied by any inability to understand spoken or written language. In the uneducated, as in children, the acts of talking and writing are closely joined, as may be seen by watching the lips, which move in the act of writing. But among those accustomed to write much these acts are independent, and it is probable that many educated aphasics may be able to answer questions in writing when their efforts at speech fail; but as a matter of experience, it is found that speech and writing are usually lost together. Reading aloud will also be lost in motor aphasia, for here, too, the inability to articulate hampers the patient.

Such patients can usually say "no" and "yes," and often retain the power of using monosyllabic words. They talk, if at all, as a baby talks who is just learning to speak. Sometimes a short phrase, uttered just before the attack of aphasia occurred, can be and is repeated over and over. Thus I knew a woman whose only phrase was "Ah, dear me, I don't know!" and this was said on all occasions, with varying inflection, being the only thing she could say.

5. Agraphia. The independence of the effort-memories, necessary for writing, from the effort-memories of speech, though questioned by Dejerine, must be admitted. When these are lost alone the condition is known as agraphia. In such a state the pen cannot be used. Copying, writing at dictation, and voluntary writing are all lost. It has been noted already that when a word cannot be called to mind, or read, or mentally enunciated, in the majority of persons, it cannot be written. But words can then often be written at dictation, if the person is one who has written much. Hence sensory agraphia and motor agraphia must be distinguished, the former being a part of word-blindness, the latter not at all associated with inability to read. The lesion of motor agraphia is not certainly known, though a few facts point to the posterior part of the second frontal convolution as the probable seat of this function. It is not unlikely, however, that the more exact localization of fine movements of the thumb and fingers in the posterior central convolution may be followed by the discovery of the writing centre in this vicinity. An interesting case is recorded by Trousseau of a deaf mute who had learned to talk by the finger language and who lost this power by a lesion near the motor centre for the fingers, though the fingers were not paralyzed.

6. PARAPHASIA OR INTERCORTICAL MOTOR APHASIA. The forms of aphasia thus far studied are due to a loss of distinct memory pictures. The several memory pictures which are united in the word-image may thus be reasonably regarded as separate from one another in their location in the brain. But, since they are joined together to form the word-image, it follows that the association-fibres joining the various areas are as necessary to the use of even a single word as the various areas with their memories. It is really by association only that an object or a word becomes a subject of thought or of use. If these associations are broken, the result is a defect of language characterized by the misplacement of words, and the patient talks jargon. Such a condition is termed paraphasia or intercortical aphasia.

There are as many forms of paraphasia as there are association-tracts.

Allusion has already been made to intercortical sensory aphasia. Another form is intercortical motor aphasia, in which the association tract between the temporal convolution and Broca's convolution is involved. This tract passes beneath the island of Reil. (Fig. **193**, D.) When it is affected the patient can understand what is heard or seen and can enunciate words clearly, but is unable to repeat after another person a word heard, and talks jargon. These patients usually talk rapidly and constantly, try very hard to convey their ideas, but cannot be understood at all, for the mingling of nonsense words or of syllables with words which they had no intention of using renders the sentences unintelligible.

If one constructs a diagram like the following figure, and then hypothetically divides one of the lines between the various centres, one obtains a scheme illustrating aphasia of conduction or intercortical aphasia. It becomes evident that there are a great many possible forms of aphasia of conduction. But the common feature in all is the loss of power of association of the memory pictures while these pictures are preserved. It is interesting to discover such cases, and much care has been given to their analysis. In fact it is largely by their analysis that our knowledge of the existence of separate memory pictures, of the localization of these pictures and of the union of them by the association-fibres has been established. But there are not as yet on record a sufficient number of cases with autopsy to enable us to make an exact diagnosis of the location of the lesion in patients who present symptoms of aphasia of conduction.

To examine an aphasic thoroughly it is necessary to test:

1. The power to recognize objects seen, heard, felt, tasted, or smelled, and their use.

This will determine whether the condition of apraxia or disturbance in the power of recalling any part of a concept is present.

2. The power to recall the spoken name of objects seen, heard, handled, tasted, or smelled.

3. The power to understand speech and musical tunes.

4. The power to call to mind objects named.

This will test the integrity of the auditory speech area and of the association-tracts between other sensory areas and the temporal convolutions.

5. The power to understand printed or written words.
6. The power to read aloud and to understand what is read.
7. The power to recall objects the names of which are seen.
8. The power to write spontaneously and to write the names of objects seen, heard, etc.
9. The power to copy and to write at dictation.
10. The power to read understandingly what has been written.

FIG. 195.

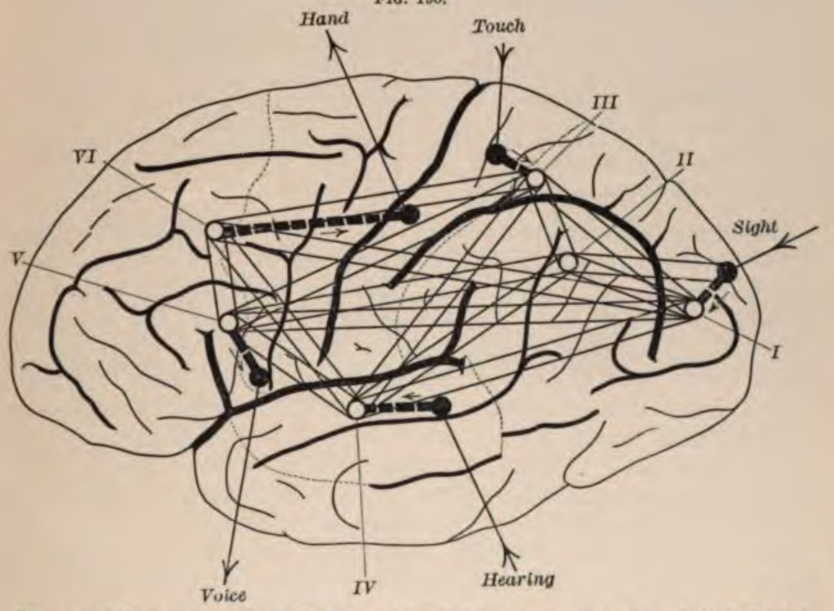


Diagram to illustrate aphasia. The cortical sensory and motor centres are indicated by the arrows. The secondary cortical centres of memories are indicated by circles. *I*, visual, of objects *II*, visual, of words; *III*, tactile; *IV*, auditory; *V*, speech; *VI*, writing. These are joined to one another by association fibres which transmit impulses in both directions. Subcortical lesions in these fibres cause aphasia as well as lesions in the cortex.

These tests will determine the condition of the visual word memories in the angular gyrus, and of the connections between this area and surrounding sensory and motor areas.

11. The power to speak voluntarily, and if it is lost, the character of its defects.
12. The power of repeating words after another.

This will test the integrity of Broca's centre and its association-tracts.

The following table shows the chief defects in each variety of aphasia, and may serve to aid in diagnosis.

Table VIII.—The Symptoms in Different Forms of Aphasia.[1]

Variety.	Understanding of language.	Power to repeat words.	Power to talk.	Power to read.	Power to write.
1. *Word-deafness.*					
(*a*) cortical,	Lost.	Lost.	Retained (not to answer).	Retained (not aloud).	Retained.
(*b*) subcortical.	Lost.	Lost.	Imperfect (not to answer).	Retained (not aloud).	Retained.
2. *Word-blindness.*					
(*a*) cortical,	Retained.	Retained.	Retained.	Lost.	Lost.
(*b*) subcortical.	Retained.	Retained.	Retained.	Lost.	Imperfect.
3. Intercortical, between T. and O.	Imperfect.	Retained.	Retained.	Imperfect (without comprehension).	Retained (not at dictation).
4. *Motor aphasia.*					
(*a*) cortical,	Retained.	Lost.	Lost.	Retained (not aloud).	Lost.
(*b*) subcortical.	Retained.	Lost.	Lost.	Imperfect (not aloud).	Lost.
5. *Agraphia.*					
(*a*) cortical,	Retained.	Retained.	Retained.	Retained	Lost.
(*b*) subcortical.	Retained.	Retained.	Retained.	Retained	Imperfect.
6. Intercortical, between T. and F.	Imperfect.	Lost.	Jargon.	Imperfect.	Lost or imperfect.

The Commissural Fibres. The second system of association-fibres in the centrum ovale is the commissural system. This joins corresponding areas of the two hemispheres with one another. The function of these fibres is to harmonize the action of the two hemispheres. Movements of like nature can be made with greater facility with both upper extremities when moving simultaneously. Movements which are difficult when attempted with the left hand alone become easy when associated with corresponding movements of the right hand—as, for example, drawing a circle, writing one's name. Such associated motions are accomplished by aid of the commissural fibres between the two motor areas.

The sensory areas are also necessarily joined by commissural tracts; for in order that the half images received in each occipital lobe may be combined, a large tract passes from one cuneus to the other. In order that sensations of touch may be correlated the two parietal lobes are joined. In order that sounds may be heard the temporal lobes are connected. The anterior commissure of the brain joins the two temporal lobes together. The commissural fibres between the greater part of the convexity of the hemispheres pass in the corpus callosum.

The existence of areas of the cortex whose chief function is to receive and transmit association impulses has recently been proven by Flechsig, and his researches throw much light upon the function of those extensive areas of the cortex which have no known sensory or motor functions. It is known that the medullary sheath which surrounds and insulates the axone of each neurone body develops after the axone. Flechsig has shown that the period at which this medullation occurs differs in different functional tracts during embryonal life. In the earliest stage but a few fibres are medullated. As the embryo grows, each system of neurones, one by one, becomes completely developed, and by contrasting brains at different ages these functional systems can be distinguished from each other.

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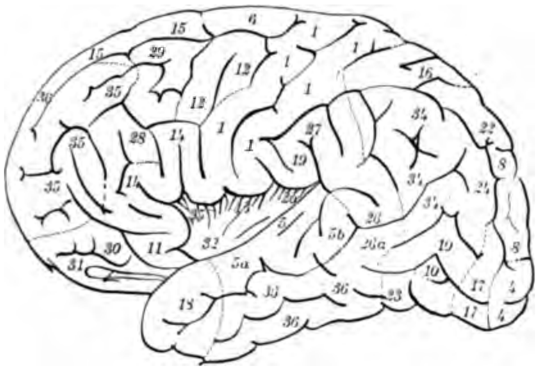
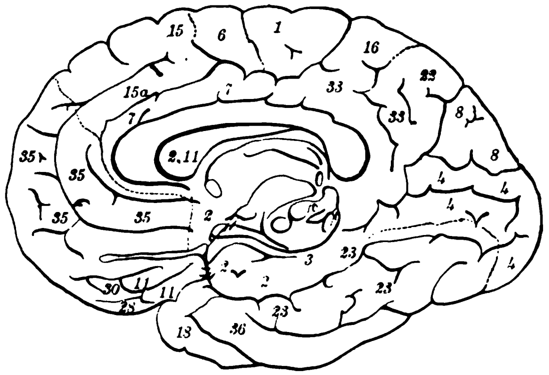
[1] There are many complex varieties of aphasia too rare and involving too exhaustive discussion to be included in a general text-book. The reader is referred to an article by the author on Sensory Aphasia, Brain, vol. xii. p. 82; to Wylie, Disorders of Speech, Edinburgh, 1894; and to Collins, The Faculty of Speech, New York, 1898, where full references to the literature of the subject are to be found.

Fig. 196.



Flechsig's diagram to show the order of development of the various areas of the cortex. Lateral surface. (Lancet, October 19, 1901.)

Fig. 197.



Flechsig's diagram to show the order of development of the various areas of the cortex. Median surface.

The chief sensory systems are the first to develop, the motor system follows, and at birth these tracts which bring the child into relation with the outer world, projecting its impressions on his consciousness and projecting his will, as shown by effort and act on the world, are complete. Later the various association-fibres within the brain are formed, so that as the sensory impressions are received they can be related to one another, and a sensation can awaken its properly co-ordinated motor response. Flechsig now distinguishes thirty-six areas of the cortex of the brain, the neurones in each area becoming developed at a different time from those in other areas. And he naturally concludes that those whose manifest function is to associate the different parts of the brain with one another, and which develop last of all, are more closely related to the higher mental process of reasoning than those whose function is merely to transmit sensations or motor impulses.

Mental symptoms consisting of aphasia, disturbances in the rapid association of ideas, apraxia, forms of loss of memory, defect in the keen perception of the meaning of ideas, defects in judgment and in reasoning, in logical thought and action—these are the symptoms that we ascribe theoretically to a disturbance of function in the association areas of the cortex or in the association and commissural tracts. And a careful clinical study of cases in which lesions have been found in the so-called latent regions of the cortex and in the centrum ovale and corpus callosum leads me to believe that such mental symptoms may always be elicited.

They will be noticed more especially in the chapters upon apoplexy, cerebral abscess, and cerebral tumors.

Disturbances in the control of the emotions, leading either to undue excitement, causeless laughter, unusual crying, great depression and a lack of harmony between the association of ideas and the state of feeling which they should awaken, are symptoms produced by lesions in the frontal area of the cerebral cortex and of the subjacent white matter of the centrum ovale. The same symptoms are also noticed in lesions of the anterior portion of the corpus callosum which unites the two frontal lobes. The frontal region is joined by a large tract to the optic thalamus. (Fig. 178, A^2.) This tract passes inward, fills the anterior limb of the internal capsule, and ends in the external nucleus of the thalamus. It was noticed by Nothnagel that lesions in the thalamus interfere with the automatic facial expression of emotion. Thus a patient who has such a lesion may not smile on the side opposite to the lesion when amused, even when he can voluntarily contract the risorii muscles, and thus give a forced smile. This is another proof of the intimate relation of the frontal region to emotional acts.

These are the chief symptoms that are met with in cerebral cortical disease and their local significance.

Symptoms of subcortical lesions. It remains to consider some special symptoms produced by subcortical lesions located in the basal ganglia and in the cerebral axis. These will be better understood because of the facts which have been already presented regarding the anatomy of the brain.

Lesions of the basal ganglia, viz., the corpora striata, made up of the lenticular and caudate nuclei and optic thalamus, are very common and give rise to many symptoms. These symptoms are, however, to be ascribed to a coincident affection of the motor and sensory tracts which pass through the internal capsule between the ganglia. (Figs. 173 and 178.) Hence, the local symptoms of lesions in the lenticular or caudate nucleus and optic thalamus are those of lesions of the various tracts in the internal capsule opposite those bodies, viz., in its posterior division. If the symptoms are permanent, the capsule is probably injured. If the symptoms pass away the capsule was incidentally affected. And the effects of the lesion may entirely subside while the lesion remains if it is entirely limited to either of these ganglia. It is therefore evident that we cannot locate a lesion in the ganglia from any direct local symptoms; for, as we are ignorant of the function of the ganglia, we do not know what is the effect of their destruction. The caudate nucleus is supposed to have some relation to motions of the legs, and the lenticular to those of the arms, their relative development in the kangaroo and bat suggesting this. The lenticular nucleus has been thought to have some relation also to acts of eating.

The hemichorea and hemiathetosis which occasionally remain after lesions of the thalamus are to be regarded as symptoms of irritation constantly exerted upon the motor or sensory tracts passing near it. There are some facts to support the assertion that the optic thalamus is a sensory ganglion, especially the fact that lesions of the pulvinar cause hemianopsia. It appears from anatomical investigations of von Monakow that all the sensory tracts end in the thalamus, which in turn is connected by its radiations with all parts of the cortex. Each sensory tract is bilateral, hence a unilateral lesion of the thalamus causes no complete loss of sensation. von Monakow has distinguished seven separate masses of neurones in the thalamus, and traced the connection of each mass to one or more distinct regions of the cortex. As yet, however, no practical diagnostic conclusions have followed that enable us to detect a lesion of the thalamus. It has also been supposed that the thalamus has some function in regulating automatic motions. Thus, in lesions of the thalamus Nothnagel found an absence of the automatic facial expression indicating emotion on the side opposite to the lesion; the patient did not laugh or cry on that side of the face, though the face was not paralyzed. But this is not always observed. Meynert saw a case, of which I also have had an example, of forced unnatural positions assumed unconsciously by the arm and leg on the side opposite to a lesion of the thalamus. Here, again, the observations are not uniform and are subject to criticism, as the capsule may have been irritated.

The basal ganglia, doubtless, have important reflex functions. The thermic centres for the regulation of the temperature of the body have been located by physiologists in the corpora striata and also in the optic thalamus, though pathology does not support this assertion. Vasomotor, secretory, and trophic control of the opposite side of the body has been a function assigned to the thalamus.

The effect of emotion on these functions is supposed to be obtained through the action of its neurones.

As a lesion limited to any one of these ganglia produces no permanent symptoms whatever, in many cases we cannot detect such a lesion. As a matter of fact, 70 per cent. of the cases of hemiplegia are due to a lesion in the basal ganglia affecting the internal capsule; and from the symptoms and nature of the disease its location there can usually be affirmed. The diagnosis is, however, made from the capsular symptoms, as already detailed.

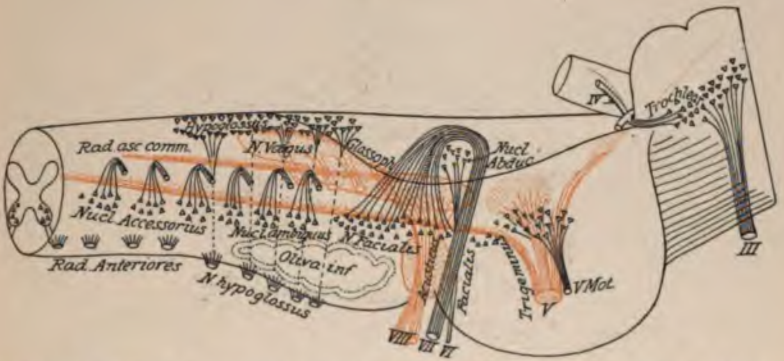
Lesions of the external capsule and of the claustrum cannot be yet located. (See Plate XVIII.) If on the left side, they usually produce paraphasia like the lesions of the island of Reil.

Lesions of the corpora quadrigemina are very rare. If the anterior pair are involved, oculomotor palsy, loss of pupil reflex, strabismus, and nystagmus may be produced. If the posterior pair are involved disturbances of co-ordination and of hearing may be caused. As both pairs are usually affected together, the combination of these symptoms may aid in diagnosis. Blindness is such a common symptom of cerebral disease that it is only when it is not due to choked disk, optic atrophy, or neuritis, and when it is not of the nature of hemianopsia, that it is to be thought a local symptom of quadrigeminal lesion, and some cases seem to show that it may not occur from a lesion there. A defective action of the same branches of the oculomotor nerves on both sides is rather more characteristic of quadrigeminal disease than the total affection of one nerve.

Lesions of the tegmentum of the crura cerebri, which lies beneath the corpora quadrigemina. Since the sensory tracts pass through this region, anæsthesia may be produced by such a lesion, and the proximity of the corpora quadrigemina will give rise to indirect local symptoms of their affection. Lesions of the red nucleus cause the same state of inco-ordination that occurs when the posterior pair of the corpora quadrigemina are involved. They also cause paralysis of the third nerve, which passes through this nucleus. Lesions of the foot of the crus cerebri, in which the motor tract passes, cause hemiplegia of the opposite side. As the third nerve issues through the foot of the crus, a lesion here causes a paralysis of this nerve on the side of the lesion. Hence, hemiplegia of one side, with third nerve paralysis of the other side, indicates a lesion of the foot of the crus cerebri on the side of the third nerve paralysis. Lesions on the base which press upon this part will produce the same combination of symptoms. (See Figs. 177 and 180.)

Lesions of the pons Varolii and medulla oblongata. Fig. 198 shows the situation in the pons and medulla of the nuclei of origin of the cranial nerves. These nuclei lie either upon the floor of the fourth ventricle, shown in Fig. 177, or at a deeper level in the formatio reticularis. From the nuclei the nerve fibres pass through the pons and medulla to make their exit upon the base of the brain, as shown in Fig. 180. It is evident, therefore, that any lesion in the pons or medulla will destroy either the cranial nerve nuclei or the nerves issuing from them and traversing the cerebral axis at the level of the lesion. The exact level of the lesion will be indicated by the nerves affected, oculomotor palsy being caused by lesions in the crus cerebri; fifth, sixth, seventh, and eighth nerve paralysis being caused by lesions in the pons, and ninth, tenth, eleventh and twelfth nerve paralysis being caused by lesions of the medulla. The various symptoms due to lesions of the cranial nerves are discussed in Chapter XXXIV., and also the means of distinguishing lesions of the nuclei from those of the nerve trunks.

FIG. 198.



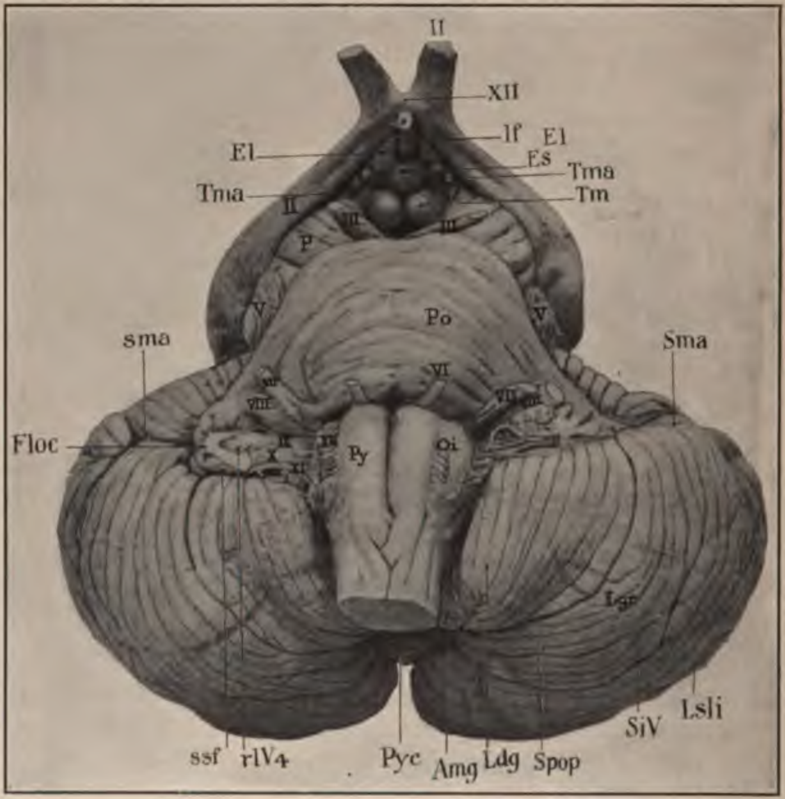
Position of the nuclei of the cranial nerves. The medulla and pons to be imagined as transparent. The nuclei of origin (motor), black; the end nuclei (sensory), red. (Edinger.)

The facts already stated regarding the various tracts passing through the pons and medulla, as shown in Figs. 177 to 181, pages 440 and 445, will be recalled. It is evident that a gross lesion like a hemorrhage or area of softening in the pons or medulla will cut those tracts and produce either unilateral or bilateral paralysis of motion or of sensation in the limbs and body. This combination of symptoms of cranial nerve paralysis and of paralysis in the limbs and body is characteristic of lesions in the pons and medulla, and leads at once to the diagnosis and localization of such lesions. The pons also contains the middle peduncle of the cerebellum, hence lesions in the pons are likely to cause symptoms of cerebellar type already considered.

Cerebellar Symptoms. Figs. 199 and 201 show the connections of the cerebellum with the cord, with the pons, and with the cerebrum. Fig. 202 shows the structure of the cerebellar cortex.

Lesions of the cerebellum, if located in the hemispheres and not in the median or vermiform lobe, and if of such a nature as not to exert pressure on surrounding parts, may not produce any symptoms. If the lesion is in the vermiform lobe, disturbance of co-ordination known as cerebellar ataxia, occurs. This consists in an inability to walk without staggering like a drunken man. The ataxia exists only while the patient is in an upright position; it rarely affects the motions of the arms, and when it does it never interferes with the fine adjustments, but only with extensive movements in space—*e. g.*, grasping objects at a distance—that involve an act to preserve the balance. Closing the eyes does not increase the ataxia. In these respects the ataxia differs from that of posterior sclerosis.

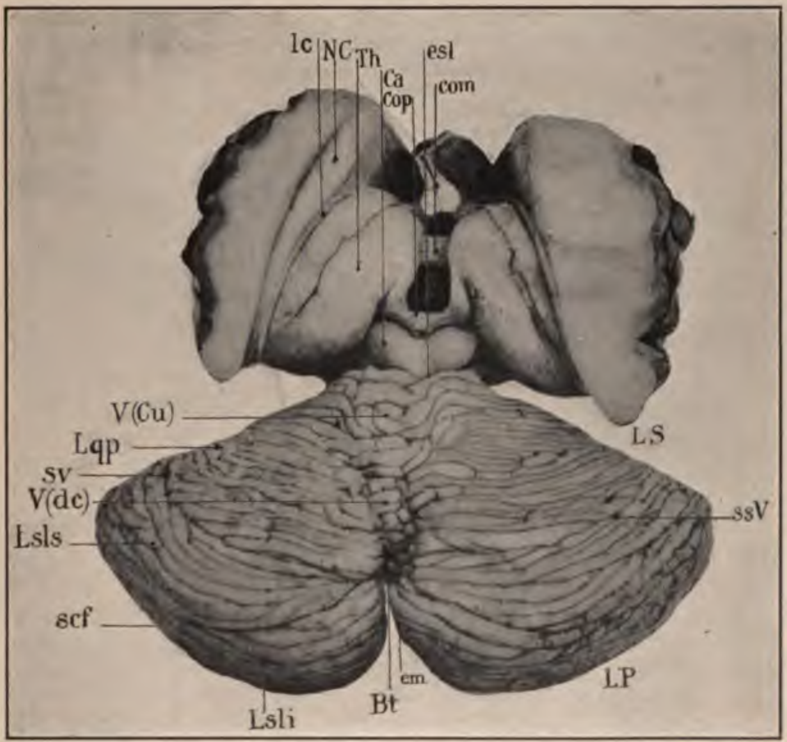
FIG. 199.



The base of the brain, the cranial nerves, and the cerebellum. II. Optic nerves. XII. Optic chiasm. If. Infundibulum. El. Lateral part of tuber cinereum. Es. Tuber cinereum. Tm. Mammillary bodies. P. Crus cerebri. Po. Pons. Py. Pyramid of medulla. Oi. Olivary body. Sma. Transverse fissure of cerebellum. Floc. Flocculus. ssf. Subfloc. fissure. rIV4. Diverticulum of fourth ventricle. Pyc. Pyramid of Malacarne. Amg. Amygdalus. Ldg. Digastric lobe. Lgr. Slender lobe. Lsli. Semilunar lobe. II. to XII. Cranial nerves. (Dejerine.)

A second characteristic symptom of cerebellar disease located in the vermiform lobe is vertigo. This may be very severe, but as it may occur without ataxia, and ataxia may be present without vertigo (though rarely), the two are not to be considered as interdependent. Vertigo is increased by rising to an erect position, but may persist when the patient is in bed. It decreases somewhat when the patient has remained fixed in any position for some time; but is always increased when he opens his eyes. The vertigo is usually an early symptom of cerebellar disease. It is more constant and persistent in cases in which the intracranial pressure is increased. It may gradually pass off in other cases. Cerebellar vertigo does not differ from vertigo in Ménière's disease, and is probably due to an affection of the terminal fibres of the labyrinthine part of the eighth nerve, from the semicircular canals or their nuclei. The vertigo of Ménière's disease is, however, usually accompanied by deafness. Ataxia and vertigo together afford strong presumption of disease in the vermiform lobe, although neither alone is sufficient for a diagnosis.

FIG. 200.



The basal ganglia and the cerebellum. Ic. Tænia. NC. Caudate nucleus. Th. Thalamus. Ca. Corp. quad. ant. Cop. Posterior commissure. Com. cent. commissure. V(Cu). Culmen. Lqp. Lobus quadratus. Sv. Fissure of Vicq d'Azyr. V(dc). Monticulus. Scf. Circular fissure. Lsli. Inferior semilunar lobe. Lsls. Superior semilunar lobe. Ls. Superior lobe. LP. Posterior lobe. Bt. Vermiform lobe, of which the culmen and monticulus are parts.

The indirect local symptoms of cerebellar disease may be numerous. They are due to an affection of the tracts and nerve nuclei in the pons and medulla (see Fig. 178). Various forms of paralysis and anæsthesia, vasomotor disturbances, obstinate vomiting of a projectile character, general symptoms of intracranial disease—*e. g.*, headache, optic neuritis—are usually present with tumors, abscesses, or hemorrhages in the cerebellum, especially if they are in the median lobe. The combination of cerebellar ataxia and vertigo with these and other symptoms of pons disease affords clear evidence of disease in the cerebellum.

FIG. 201.

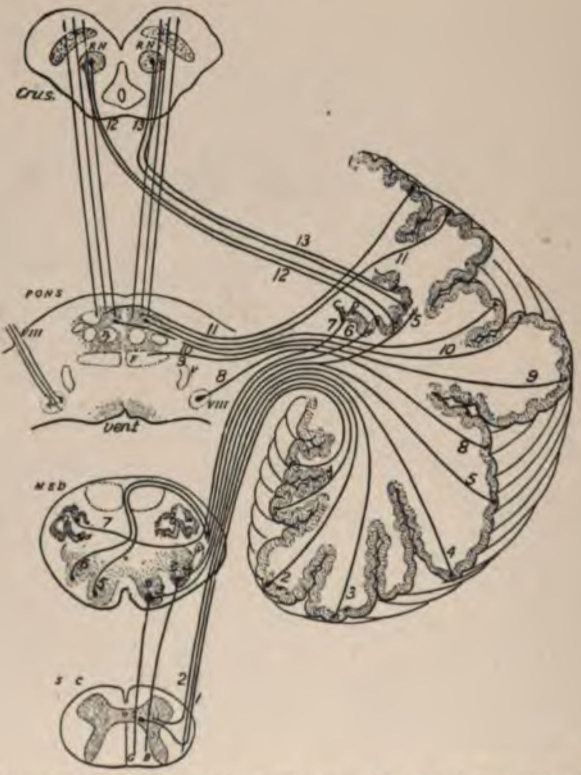


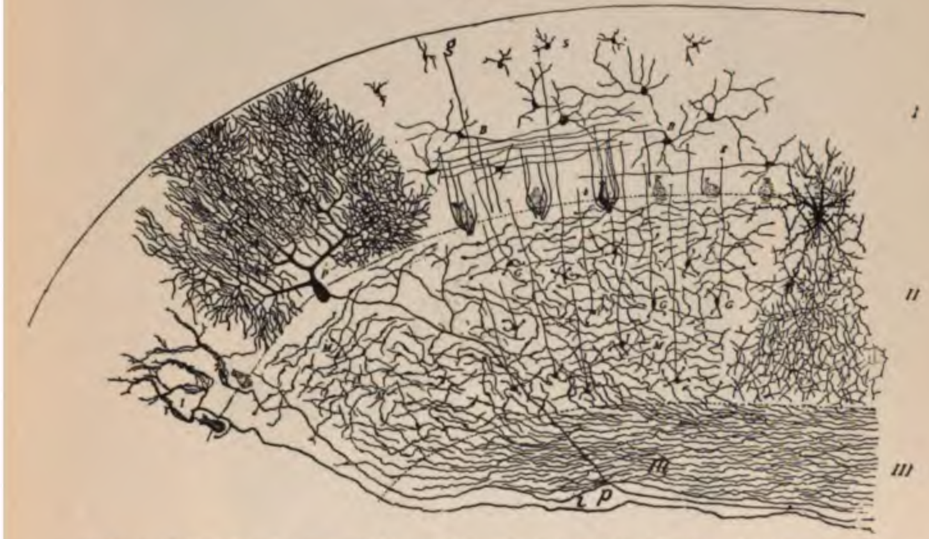
Diagram to show the connections of the cerebellum. *SC.* Spinal cord. *G.* Column of Goll. *B.* Column of Burdach. *MED.* Medulla oblongata at sensory decussation. *O.* Olive. *G.* nucleus gracilis. *C.* Nucleus cuneatus. *V.* Fifth nerve. *Pons.* Pons Varolii. *VIII.* Eighth nerve and its nucleus. *F.* Fillet. *Py.* Pyramids. *Crus.* Crus cerebri. *RN.* Red nucleus of tegmentum. *CD.* Corpus dentatum of cerebellum. 1 to 13. Various tracts connecting the cerebellum with the spinal cord, medulla, pons, and crus. (Starr, Atlas of Nerve Cells.)

Lesions of the cerebellum have no apparent effect upon the mental powers when they occur in adults. A deficient development of the cerebellum is, however, a frequent cause of congenital idiocy. When one cerebellar hemisphere fails to develop, the opposite olivary body in the medulla, and sometimes the opposite hemisphere of the cerebrum, present an atrophic appearance.

Fig. 203 shows a case of such defective development. The patient was a half-witted boy who died of measles at the age of three years. He had been unable to learn to walk, and was awkward in his movements.

Lesions of the middle peduncles of the cerebellum, the crura cerebelli ad pontem, produce characteristic symptoms. These consist in a tendency on the part of the patient to assume a forced position, to turn toward or fall toward one side in walking, or even to revolve constantly about one axis of his body. The forced movements may be made by the eyes (conjugate deviation in one direction), by the head, or by the entire body. In a case seen by the writer, in which the autopsy showed a tuberculous tumor in the left middle peduncle and in the vermiform lobe, in addition to ataxia, vertigo, vomiting, and headache, the patient lay constantly on his left side, and when he turned upon his back or toward the right side the vertigo became so excessive that he was obliged to resume at once his former position. In walking, this patient showed a tendency to fall toward the left side, and found it impossible to turn

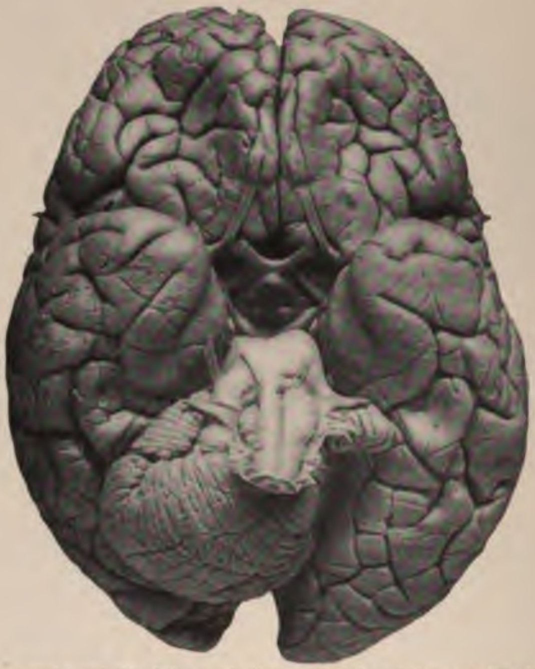
FIG. 202.



Diagrammatic representation of a section through the cerebellar cortex. *I.* Molecular layer *II.* Granular layer. *III.* White matter *P.* Purkinje cell with its neuraxone, *p*, entering the white matter. *S.* Small stellate cells of molecular layer. *B.* Large stellate cells with basket fibres, *b*—these basket fibres surround the body of the Purkinje cell shown in dotted outline. *G.* Cells of the granular layer, with long, straight neuraxone, *g*, ascending to molecular layer, and there bifurcating to become tangential fibres—these fibres run at right angles to the plane of section of the plate. *M.* Moss-like termination of white fibres, *m*, entering the cerebellum from without. *H.* Large Golgi cell of the second type, with dendrites in both granular and molecular layers and neuraxone dividing and subdividing in the granular layer. *I.* Terminal filaments and fibres, *i*, entering the cerebellum from without and ending around the branches of the Purkinje cells. (Starr, Atlas of Nerve Cells.)

around toward the right. In another case, a laceration of one peduncle due to a fracture of the base, caused constant vigorous movements of rotation about the long axis of the body toward the affected side for three days until death occurred. Such patients may lose their balance in moving in one direction—*e. g.*, forward or backward, and in attempting to regain it they may be obliged to hasten their movements. This has been interpreted wrongly as a tendency to compulsory walking in one direction—*e. g.*, backward. It is really due to the vertigo. Lesions of the other peduncles of the cerebellum do not produce any known characteristic symptoms aside from those of cerebellar disease. But when the superior peduncles are the seat of a lesion it is not uncommon to have oculomotor paralysis, especially paralysis of the fourth nerve, as an associated symptom. And when the inferior peduncle is affected there is usually an alternating hemianæsthesia from lesion of the formatio reticularis.

FIG. 203.



Congenital absence of one-half of the cerebellum. Atrophy of the opposite olivary body and of the superficial transverse fibres of the pons. (Kindness of Dr. Oliver S. Strong.)

CRANIO-CEREBRAL TOPOGRAPHY.

The diagnosis of local lesions in the brain leads in many cases to surgical treatment for the removal of the disease, such as clots, tumors, or abscesses. It is, therefore, essential to know the exact relation between prominent parts of the skull and the fissures and convolutions of the brain. This relation has been carefully determined and certain rules have been laid down.

The relation of the brain to the skull is shown in Fig. 205.

The rules for finding the fissures of Sylvius and Rolando by measuring the skull are as follows:

FIG. 204.

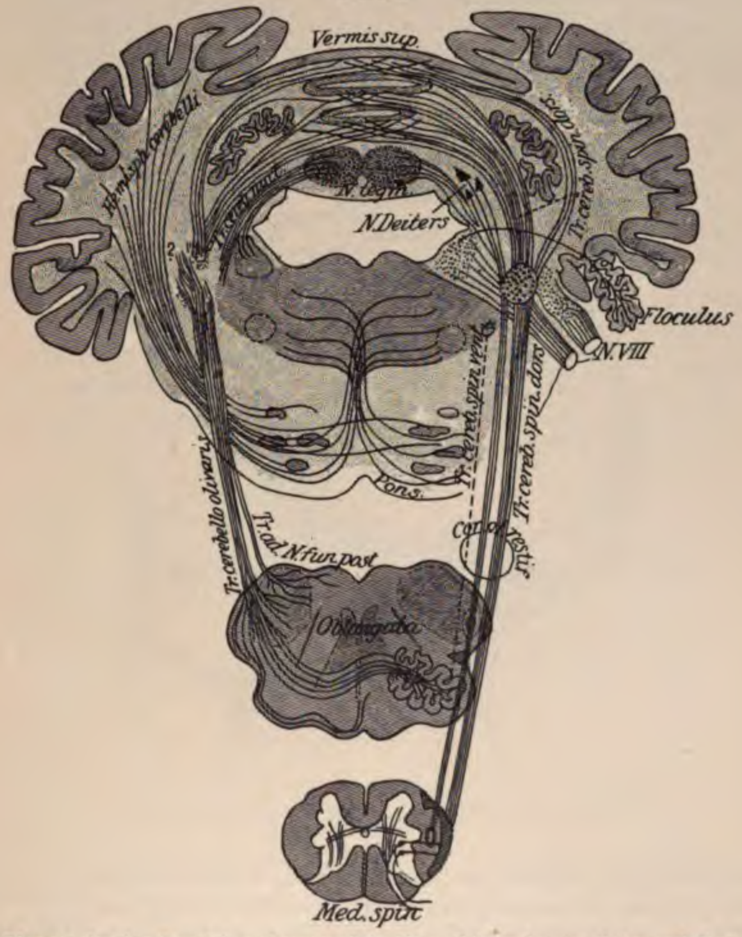
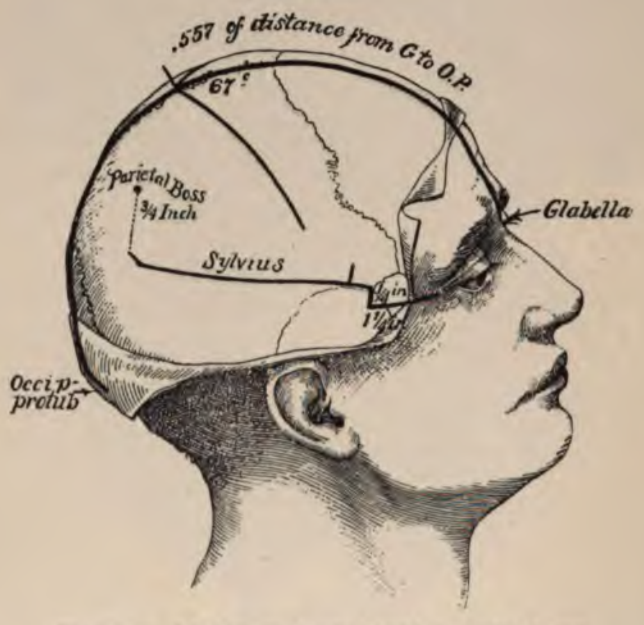


Diagram showing the origin and course of the fibres of the peduncles of the cerebellum. (Edinger.)

To find the fissure of Rolando, lay down a line from the root of the nose to the occipital protuberance over the top of the head, and take a point 0.557 of the distance back upon this line. This point will correspond to the upper end of the fissure. The fissure makes an angle of 67° with the median line just measured. Hence if two strips of metal, fixed to one another at this angle, be placed on the head with their junction upon the upper end of the fissure, when one strip is on the median line the other strip, pointing forward and downward, must lie over the fissure of Rolando. In its lower third the fissure becomes a little more vertical than the strip. The fissure is about three and a half inches long.

To find the fissure of Sylvius, lay down a base line from the lower margin of the orbit to the auditory meatus. Lay down a second line parallel to the base line from the external angular process of the frontal bone backward one inch and a quarter, and then measure upward one-quarter of an inch; this gives point one. Find the most prominent part of the parietal eminence, and from it draw a line downward perpendicular to the base line, and on this take a point three-quarters of an inch below the eminence; this gives point two. Join these two points, and the line will lie over the fissure of Sylvius. The anterior limb of the fissure will be two inches behind the external angular process. The fissure of Sylvius is about four inches long.

Fig. 205.



The relation of the fissures of Rolando and Sylvius to the skull.

To find the parieto-occipital fissure, continue the line of the fissure of Sylvius to the median line. At their junction lies this fissure.

Since all areas now open to surgical operation can be located with a definite relation to these three fissures, no further rules are necessary. As in opening the skull it is customary to make a fenestrum of at least an inch in diameter, and as it is frequently necessary to enlarge the opening much more, a procedure in no way dangerous under aseptic conditions, there is no difficulty in recognizing the fissures and convolutions exposed if the rules are closely followed. Prior to the large incision of the scalp it is well to mark certain points upon the skull by the sharp point of a chisel, so that when the bone is laid bare surface landmarks may still be kept in view.

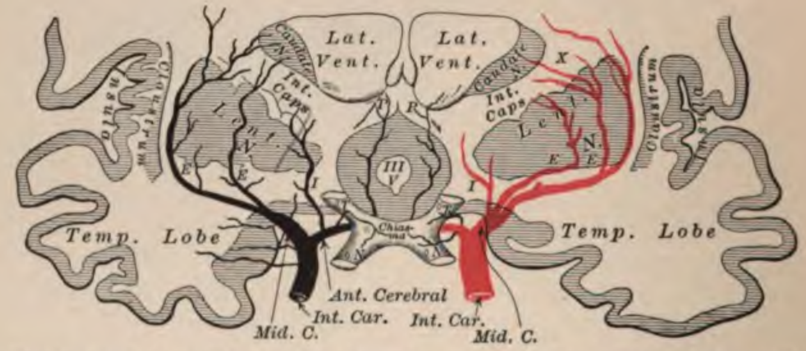
CHAPTER XXV.

THE CEREBRAL CIRCULATION.

THE majority of cases of brain disease which are met with in practice are due primarily to disease of the bloodvessels. Endarteritis causes thrombosis and embolism, and, also, by the final weakening of the vessel wall, rupture and hemorrhage. In the former case necrosis of the brain tissue is produced, with arrest of its functions. In the latter case laceration and destruction of the brain tissue is caused with both irritation and arrest of function. Hence a knowledge of the distribution of the cerebral bloodvessels is a necessary preliminary to a study of the various types of apoplexy.

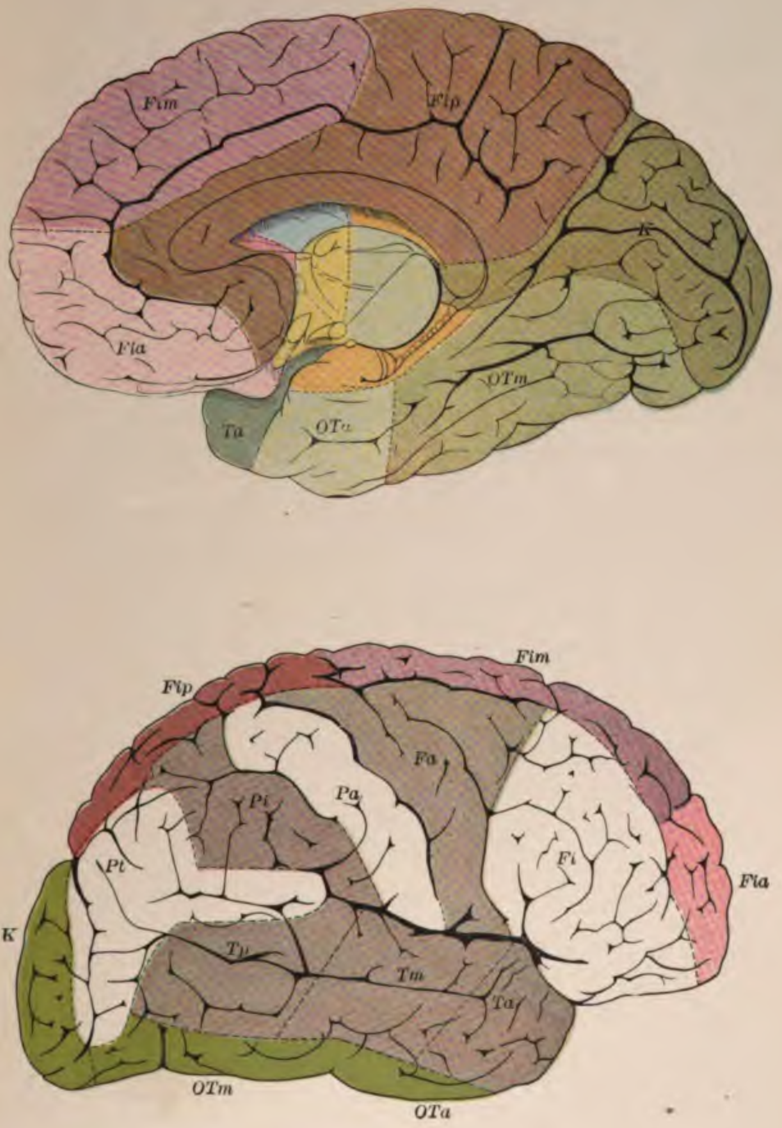
The blood reaches the brain through the vertebral and internal carotid arteries. The vertebral arteries unite to form the basilar, which in its course gives off the arteries of the cerebellum and pons. The basilar divides into the posterior cerebral, which supply the thalamus, the tegmentum of the crura, the corpora quadrigemina and crura, and the temporo-occipital basilar part of the hemispheres. The posterior cerebral send the posterior communicating arteries forward to join the internal carotid. These too send small vessels into the basal ganglia. The internal carotid divides into the middle and the anterior cerebral, the latter being united by the anterior communicating, which thus completes the circle of Willis. From the arteries composing this circle many little branches perforate the base of the brain supplying its basal ganglia.

FIG. 206.



Showing distribution of bloodvessels to internal capsule. The artery marked x is the so-called artery of cerebral hemorrhage, and it is readily seen that its rupture destroys the fibres in the internal capsule. (Duret.)

PLATE XX.



The Vascular Supply of the Cerebral Cortex. (Dejerine.)

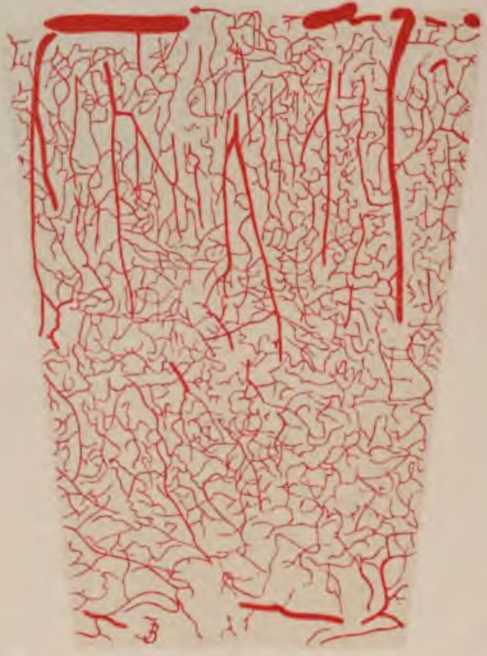
The regions supplied by different arterial branches are shown in different colors.

The median surface.—Branches of the anterior cerebral artery. *Fia.* Anterior frontal. *Fim.* Middle frontal. *Fip.* Posterior frontal. Branches of the posterior cerebral artery *OTa.* Temporo-occipital anterior. *OTm.* Temporo-occipital median. *K.* Calcarine.

The lateral surface.—Branches of the middle cerebral artery. *Fi.* Inferior frontal. *Fa.* Ascending frontal. *Pa.* Ascending parietal. *Pi.* Inferior parietal. *Pc.* Angular. *Ta.* Anterior temporal. *Tm.* Middle temporal. *Tp.* Posterior temporal.

PLATE XXI.

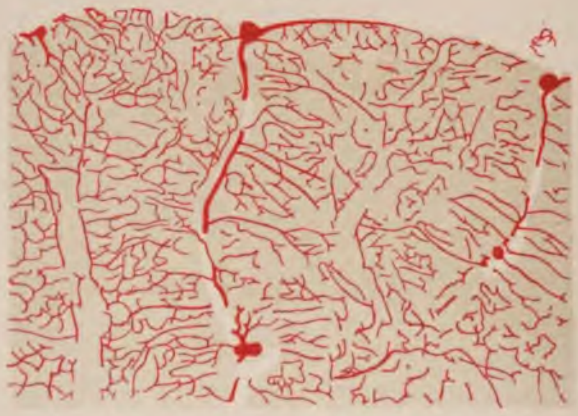
FIG. 1.



The Arterial Supply of the Cortex Cerebri. (Szymonowicz.)

Section through the cerebral cortex of a rabbit; bloodvessels injected red. × 40.

FIG. 2.



The Arterial Supply of the Cortex Cerebelli. (Szymonowicz.)

Section through the cerebellar cortex of a guinea-pig; bloodvessels injected red. ×44.

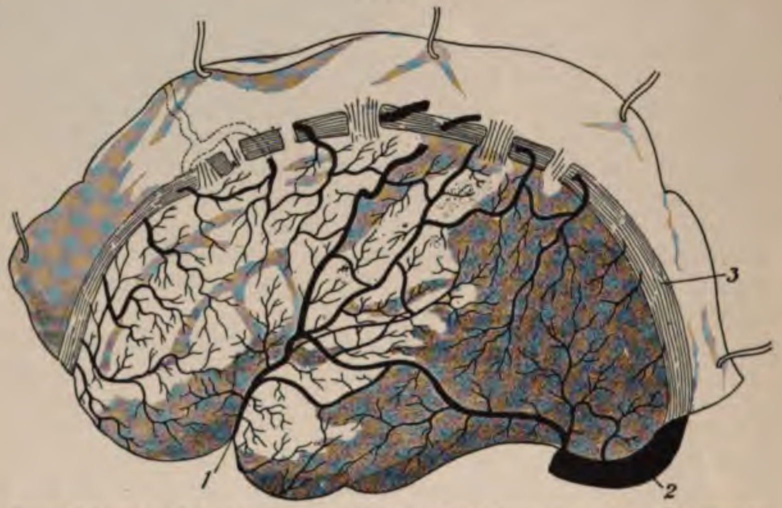
The middle cerebral artery, passing outward through the fissure of Sylvius, gives off in its course many basal arteries which enter the anterior perforated space. These ascend through and outside the lenticular nucleus and supply the internal capsule and the anterior part of the optic thalamus. (Fig. 206.) The middle cerebral artery divides as it passes outward into several branches which radiate over the island of Reil, and spread outward over the convexity of the brain supplying the greater part of the lateral surface. (Plate XX.) The larger branches go to the third frontal convolution, to the anterior and posterior central convolutions, and along the fissure of Sylvius. The Sylvian artery gives off numerous branches to the operculum and supramarginal convolution above and to the temporal convolutions below, and finally terminates by spreading out over the parietal lobules and the convolutions joining the temporal and occipital regions.

The anterior cerebral artery passes forward, supplying the base of the frontal lobe, then curves upward on its median surface, sending numerous branches over the edge. It supplies the median surface of all the frontal convolutions and a small part of the cortex of the lateral surface near to the median line, excepting the third, which receives its supply from a branch of the middle cerebral. Its terminal branches pass backward as far as the cuneus, anastomosing on the way with the terminals of the middle cerebral artery, and with those of the posterior cerebral artery.

The anastomoses of the cortical terminal vessels are most perfect, as shown in Plate XXI. But many of the small basilar vessels have few or no anastomosis with adjacent branches, and hence have been called terminal arteries. The effect of an embolism in such arteries is to cause an area of necrosis which is permanent. The basal arteries do not anastomose freely with the cortical arteries, and hence the white matter lying above the basal ganglia and beneath the cortex is relatively imperfectly supplied with blood. This protects it from the occurrence of hemorrhage, but predisposes it to softening after thrombosis or embolism. The cerebellum is supplied by blood-vessels which arise from the vertebral, basilar, and posterior cerebral arteries. The blood supply of the crura cerebri and pons and medulla is derived from branches of the basilar and posterior communicating arteries.

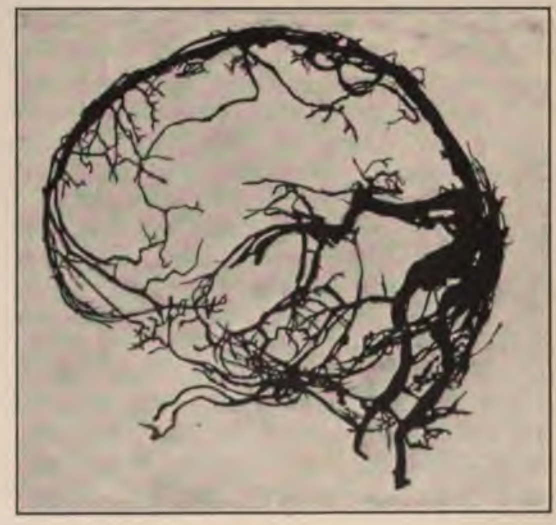
It is evident that the effect of an embolism or thrombosis in any of these vessels may be a very small or an extensive necrosis of tissue, according to the size of the vessel plugged. The extent of a hemorrhage in the brain will also depend on the size of the vessel ruptured. There are certain vessels in which embolism and hemorrhage are more common than in others. The small vessels, which enter the base through the anterior perforated space come off from the upper surface of the internal carotid artery or the middle cerebral. The current of blood rushes directly into these, as it is forced up from the heart more forcibly than into other vessels, to reach which it has to pursue a more tortuous course. Hence, these are the vessels more commonly ruptured under an increase of pressure or plugged by a clot or embolism from the heart. The distribution of these vessels is to the basal ganglia and internal capsule. Hence the frequency of

FIG. 207.



Superficial veins of the external surface of the left hemisphere. 1. Great anastomotic vein of Froland. 2. Lateral sinus. 3. Longitudinal sinus. (Testut.)

FIG. 208.



An injected and corroded preparation of the cerebral veins and sinuses. (Huntington.)

capsular lesions. The terminal character of these vessels makes repair impossible. The free anastomosis of the cortical vessels has already been noticed. A thrombosis or embolus in one of them rarely has a permanent effect, as the necrosis at first begun is arrested by the development of a collateral supply of blood. Hence cortical lesions have a more favorable prognosis than capsular lesions.

The venous circulation in the brain is shown in Figs. 207 and 208. The veins from the interior of the brain empty into the veins of Galen and then into the straight sinus. Those from the cortex reach the longitudinal, lateral, and cavernous sinuses.

CHAPTER XXVI.

THE CEREBRAL DISEASES OF VASCULAR ORIGIN.

CEREBRAL HYPERÆMIA AND ANÆMIA.

THE investigations of Mosso have proven that any mental effort or emotion is attended by a functional hyperæmia of the brain and that sleep is attended by a functional anæmia.

Formerly a number of abnormal conditions were ascribed to *hyperæmia of the brain*. It is doubtful, however, if they were dependent upon this cause, and it is improbable that congestion of the brain alone is a condition that can be more than temporary or that can produce serious permanent symptoms. It is admitted that in many constitutional states both active and passive cerebral congestion may occur. Thus fever, persistent mental strain, great emotional excitement, sunstroke, and an excess of alcohol, quinine, and strychnine produce congestion of the brain. Obstruction to the flow of blood from the cerebral sinuses may lead to venous congestion. In this condition the symptoms of the original underlying disease, such as tumor or abscess of the brain, thrombosis of a sinus, or tumors and aneurisms in the neck, chronic cardiac disease, emphysema, etc., will obscure any symptoms due to the hyperæmia of the brain. There is doubtless a stage of congestion of the brain in all cases of meningitis and of encephalitis, but here again it is not possible to distinguish between the symptoms due to the two conditions. The symptoms produced by congestion of the brain are headache of a severe character, with feelings of pulsation, vertigo increased by a sudden effort or change of position, tinnitus aurium, sensations of pressure about the eyes and head, insomnia, mental irritability, with sense of incapacity to carry on a train of thought, and restlessness. The convulsions and coma in sunstroke are due to toxæmia and not to the congestion. It is probable that the majority of the symptoms mentioned may, in some cases, be due to imperfect metabolism, or to chemical changes in the nerve cells, rather than to mere mechanical pressure. I am always reluctant to ascribe morbid states to congestion of the brain, and I regard such a diagnosis as unsatisfactory and tentative. I do not believe in the existence of a chronic congestion. The treatment of the symptoms during their temporary duration is by ice to the head and the free use of ergot and bromide of sodium. Such treatment will relieve the headache and sensations of pressure in the course of a few hours, but should not be kept up more than two or three days. It is well to give calomel, followed by saline laxatives, to restrict the diet, to cut off all alcoholic stimulants, and stop the use of tea, coffee, and tobacco; in other words, to eliminate from the body all those poisons which may be the cause both of the congestion and of the symptoms. If the cause of venous congestion can be ascertained it is to be removed, if possible. If impossible, wet cups behind the ears, hot foot baths, counter-irritation to the neck, and sedatives may relieve the symptoms.

Anæmia of the brain may be due to loss of blood or to a general anæmic condition. The latter is not uncommon both in young girls and in women at the menopause as a primary disease. It may also be secondary to other constitutional or organic diseases that produce malnutrition. The symptoms of anæmia of the brain due to a loss of blood are faintness, vertigo, nausea, confusion of ideas, and finally a loss of consciousness, with suspension of respiration and great feebleness of the heart. They are usually relieved soon by a recumbent posture, by inhalation of ammonia, and by stimulation with alcohol. In extreme cases transfusion with salt solution may be required.

The symptoms of anæmia of the brain due to general anæmia are headache, felt chiefly on the vertex, somnolence, especially after meals, depression of spirits, inability to concentrate the attention, or to conduct any active mental process for any length of time, and insomnia. The insomnia of anæmia is characteristic. The patient gets to sleep easily on lying down, but sleeps only a short time, and then wakes and is unable to get to sleep again.

The treatment of the symptoms is by stimulation of the heart, but this should be temporary only. The underlying state is the only proper object for treatment. Hence food, iron, arsenic, beef marrow, and tonics of all kinds are indicated. In anæmia hydrotherapy and massage are of great value.

APOPLEXY.

THE most common cerebral diseases are those which are due to a rupture of a bloodvessel in the brain, *cerebral hemorrhage*, or to a stoppage in a cerebral vessel, *cerebral embolism*, and *cerebral thrombosis*.

Pathology. The condition which leads to such diseases is chronic endarteritis. This is a chronic inflammatory process in the intima and muscular coats of the vessels, with a production of new cellular tissue, which may (1) increase until it obliterates the lumen of the vessel, producing thrombosis; or, (2) undergo calcareous degeneration, roughening the wall and inducing fibrin deposits, which may occlude the vessel, producing thrombosis, or be washed onward, producing emboli [illegible], (3) undergo fatty degeneration, thus eroding the [illegible] ening the muscular coat, causing aneurisms [illegible] ch r [illegible] roducing cerebral hemorrhage. [illegible] smaller in diameter, and are [illegible] space or have little support.

While they are usually due to endarteritis, von Monakow believes that they may form in senile arteries by a bulging of a normal wall the muscular coat of which is merely thinned but not diseased.

The causes of endarteritis are syphilis, the abuse of alcohol, chronic poisoning by lead, the development of poisons within the body as the result of imperfect nutrition, or of indigestion due to too much food and too little exercise, gout, nephritis, and old age. The affection is distinctly hereditary, and in many families death from apoplexy is the rule. The use of tobacco, coffee, and tea is said to predispose to endarteritis, but this is not established.

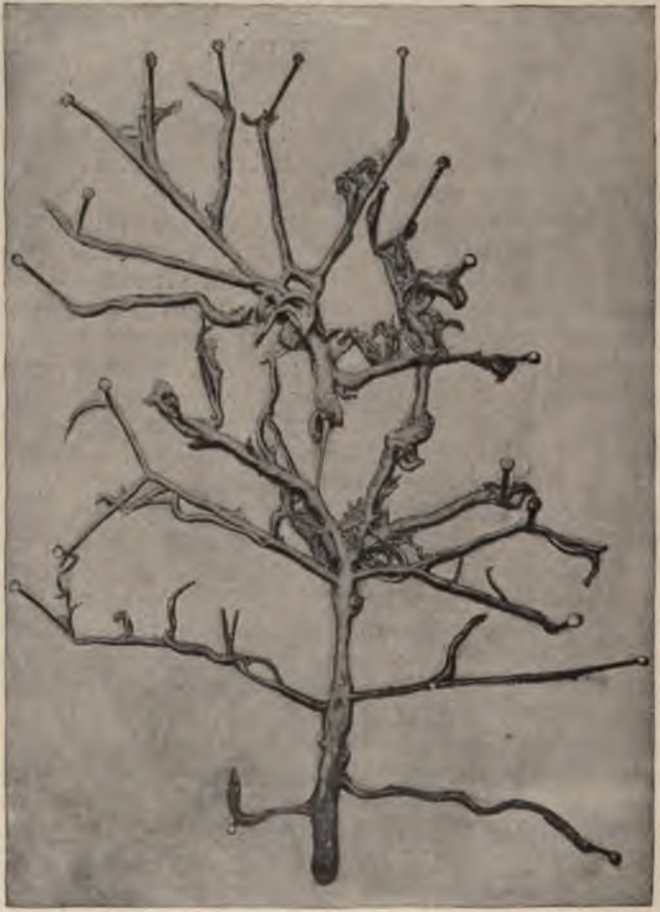
Pathology of Cerebral Hemorrhage. When a cerebral vessel ruptures in the membranes the blood spreads out on the surface of the brain, filling the fissures and depressing the cortex. The clot which forms is thin and flat. It may become organized and adherent to the pia mater, or it may lie as a clot for weeks. In one case in which McBurney operated for the removal of such a clot from the third frontal convolution, for the relief of aphasia, three months after the hemorrhage the clot was only partly organized and was easily sponged off from the surface and from the fissures. If the hemorrhage is subcortical it tears the surrounding brain, sometimes breaking into the ventricles and filling them with blood. It may be a small clot or it may be almost as large as the hemisphere, disintegrating its greater portion. Usually it is about two inches in diameter. The clot is globular or irregular in shape in the brain, or forms a cast of the ventricles. It compresses the brain tissue around it, and, as a rule, a number of capillary hemorrhages are found in the adjacent brain. The adjacent brain is always œdematous. The clot gradually shrinks, and if small undergoes absorption, leaving a hematin stain. The disintegrated brain tissue undergoes fatty degeneration, is liquefied, and is in part absorbed. There remains a mass of softened tissue, mingled with blood, which after a time becomes organized by the formation of connective tissue in and about it. This connective tissue may form a thick layer, or even a firm fibrous plaque, upon the surface of the brain, closely adherent to the cortex, and supplied with numerous bloodvessels. In one case operated upon two years after an injury, with hemorrhage, it was as hard as cartilage. If it is within the brain a firm mass of connective tissue enclosing the softened mass may grow. Then, as the softened mass within this wall becomes liquefied, the final result is a cyst filled with fluid, either bloody or clear, and serous. In one case I saw a connective-tissue structure resembling a honeycomb, its cavities filled with clear fluid. A clot begins to lose its bloody color from the second to the fifth week, depending on its size; after the eighth to the twelfth week it is a yellowish mass with hematin stains, and within three to six months the connective tissue formation is well under way. This contracts during a year after its formation, and the process cannot be said to have reached its final stage of scar tissue or cystic formation until the end of the second year. A cyst wall may be apparent within a month of the onset in small hemorrhages, encapsulating the softened mass. The torn brain never unites. It undergoes a fatty degeneration in the vicinity of the clot, and a process of secondary degeneration begins at once in the course of the tracts which are injured. By the end of the week such secondary degenerations can be detected by the Marchi stain at long distances from the original lesion, and after six weeks a sclerosis or replacement hyperplasia of connective tissue has begun in the entire length of the tract which is degenerated. These degenerations are very extensive through the centrum ovale, as they affect all the association and commissural fibres which are ruptured. They can also be traced through the projection fibres downward into the spinal cord or upward to the cortex. Figs. 33 and 34 show such descending degeneration in the spinal cord after hemorrhages of small and of great extent in the internal capsule.

The clot exerts considerable pressure upon the brain tissue about it, not only at the time of its formation but for some time after. This pressure is quite sufficient to suspend the function of the compressed brain, and may even be sufficient to set up degenerative processes. The brain tissue which is merely compressed will resume its function when the pressure is removed. Therefore, the immediate and temporary effects of a cerebral hemorrhage are usually much more severe and extensive than the permanent ones. And hence a gradual and pretty considerable relief from the initial symptoms is to be looked for in cases of cerebral hemorrhage. Such a relief, due to slow absorption of the clot, is necessarily much slower than the relief which occurs after embolism or thrombosis, where the collateral circulation is established within a week.

The situation of a cerebral hemorrhage depends upon the fact that certain arteries of the brain are particularly liable to the formation of miliary aneurisms. According to Durand-Fardel, in 75 per cent. of the cases these aneurisms are found on the small branches of the middle cerebral artery that enter the anterior perforated space, viz., the lenticulo-striate and lenticulo-thalamic branches. These arteries are fairly large branches; they are almost in a vertical line above the heart, the blood rushes in from the internal carotid artery under high pressure, and they are terminal arteries with no collateral circulation. Hence they have been termed by Charcot the hemorrhagic arteries of the brain, and as a matter of fact more than 60 per cent. of cerebral hemorrhages are due to their rupture. A clot thus forming destroys the lenticular or striate nucleus, or the optic thalamus, and the internal capsule which lies adjacent to them, or if it forms higher up and nearer the terminal branches it lies in the centrum ovale. In either case if the hemorrhage is large it is liable to break its way into the ventricle.

The Sylvian artery and its branches have little support to their walls, as they lie upon and not in the brain, and are also prone to the development of miliary aneurisms. Cerebral hemorrhage is not uncommon in the Sylvian fissure or on the cortex, being found there in 24 per cent. of the cases. The remaining 16 per cent. of the cases are divided between the brain axis and the cerebellum, the last-named organ being most rarely affected. In about 20 per cent. of the cases the hemorrhage breaks through into the ventricles, or occurs originally from a ventricular bloodvessel.

FIG. 209.

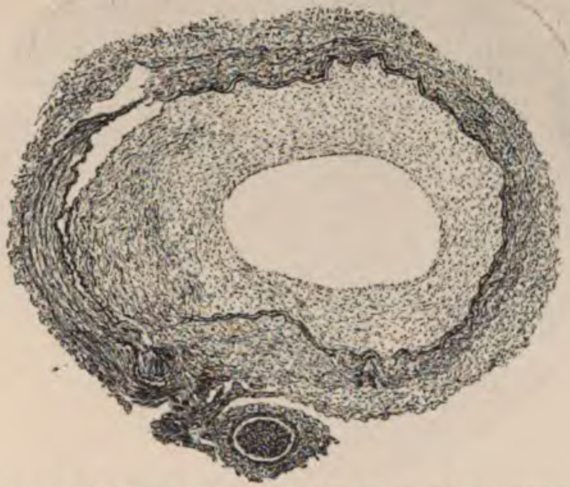


Endarteritis obliterans. Occlusion of the right posterior communicating artery of the circle of Willis. The vessel has dwindled to a fibrous cord. Foci of atheromatosis are noticeable on the basilar artery. M., aged forty-one years; case of pseudodementia paralytica uræmica. Reproduced from a photograph. (Berkeley, Mental Diseases.)

A cerebral hemorrhage may result from a diapedesis of blood cells from numerous capillaries, without rupture of the walls. The blood then collects about the vessels and infiltrates the brain compressing individual neurones and axones, but not producing large clots or cysts. The condition resulting is described by the French as hemorrhagic softening (*ramollissement hemorrhagique*). It is present in many cells in the brain tissue about a large clot. It is the condition which occurs in purpura, in pernicious anæmia, and in leucocythæmia.

Pathology of Cerebral Embolism or Thrombosis. The artery being occluded by an embolus coming from the heart or larger arteries, or by a clot forming upon the roughened wall, or near a constriction formed by obliterating endarteritis, the brain tissue which is supplied with blood through it is cut off from its nutrition. The first arrest of blood supply is usually extensive, but when collateral circulation is established the final area or region which softens may be small. The sudden stoppage of circulation in the artery and in its corresponding veins suspend the functions in this extensive area, but as the collateral circulation is resumed the functions reappear in all but the parts which are permanently deprived of blood. In the cortex the collateral supply by anastomosing vessels is extensive. In the ganglia and capsule it is very imperfect, as the arteries are terminal. Hence the permanent effect of occlusion is more serious in lesions of the arteries of the base than in those of the branches in the cortex. If a large vessel in the cortex—*e. g.*, a main branch of a Sylvian artery, or the middle cerebral trunk itself—is plugged, the area of softening may be extensive. When a vessel is occluded a clot forms within it which extends backward to the next large branch. In some cases a second embolism is produced by a portion of this clot being swept off into this branch; then a second attack follows the

FIG. 210.

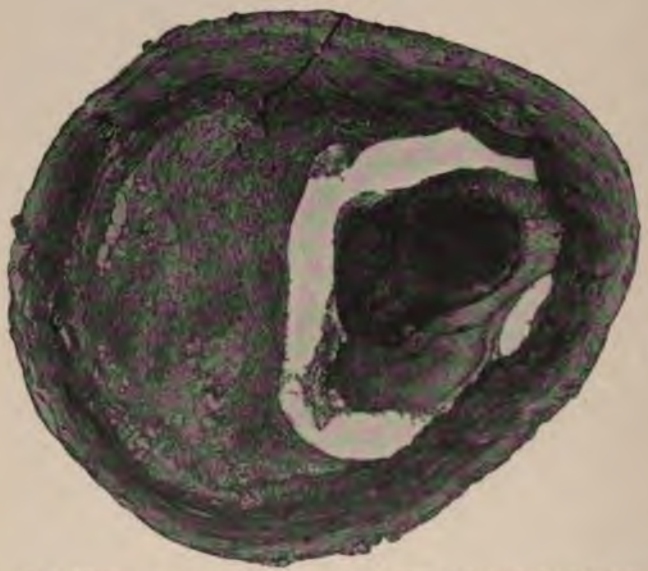


Transverse section of the middle cerebral artery from a case of obliterating endarteritis. The proliferation of the inner coat and the reduction of the lumen of the vessel are evident. (Spiller.)

first within twenty-four hours. This is the explanation of the recurrence of symptoms which have apparently subsided on the day after the attack in cases of embolism. Thus in one of my patients three attacks occurred on three successive weeks; the first caused paralysis of the arm, the second of arm and leg, the third of the entire side and face.

A serous infiltration and œdema of the brain occurs in the entire domain of the artery which is occluded, within an hour; but this is to some extent relieved when the collateral circulation is established. Capillary hemorrhage or diapedesis of blood cells is also found in the œdematous brain. There are some cases of apoplexy in which the only lesion found after death is an œdematous state of one hemisphere. These are the cases in which a thrombosis of one internal carotid or middle cerebral has been followed by a rapidly fatal termination before softening has been produced.

FIG. 211.

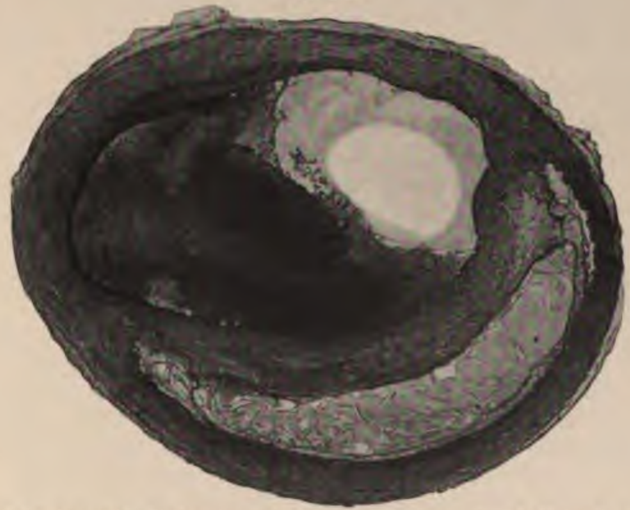


Obliterating endarteritis. The intima is greatly thickened, and one-half of the lumen is filled by deposit of newly-organized tissue that has undergone fatty degeneration in its outer part. A thrombus is seen adherent on one side to the intima. It filled the vessel, but contracted during the process of hardening the specimen.

The pathological process in cases of embolism in which the embolism is of bacterial origin, as in ulcerative endocarditis, or in pyæmic emboli, is somewhat different, as there is an acute encephalitis of limited extent set up in the necrotic area. In addition to the fatty degeneration there is an infiltration with leucocytes and the production of pus in which micro-organisms are found. In such cases there is a tendency of the process to extend beyond the limit of the tissue first deprived of its blood and to the production of an extensive encephalitis.

A fatty degeneration and a necrosis of brain tissue follow the occlusion of the vessel on the third day. In the softened tissue there are at first extravasations of blood and pigment granules (red softening); later, after four weeks, these are absorbed and the tissue in a stage of fatty degeneration is yellow (yellow softening). If there is simple necrosis without extravasation or fatty degeneration in the white matter, the brain has its natural color, but is soft (white softening). Round granule cells are everywhere to be found in this softened tissue, and are mingled with drops of myelin in all stages of fatty degeneration. The perivascular spaces are filled with leucocytes and small cells.[1] The softened tissue undergoes liquefaction or becomes organized by a connective tissue growth. This is formed partly from hyperplasia of the glia and partly from the connective tissue of the smaller vessels which proliferates. It contracts, leaving a depression, or a cavity, or a scar. In some cases a cyst with smooth walls and serous contents is formed. Such a small cyst is shown in Fig. 240, in the midst of a diffuse tumor of the optic thalamus. The size of the area of softening and of the subsequent scar may be very small, so that at the autopsy a mere patch of sclerosis is found. On the other hand, the softening may involve almost an entire hemisphere.

FIG. 212.



Obliterating endarteritis. Thickening of all the coats of the artery. The intima has encroached upon the lumen. The new tissue has undergone fatty degeneration, and a considerable mass of fatty débris in which cholesterin crystals can be seen, lies between it and the media. A thrombus is seen adherent to the intima.

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[1] See Friedmann, Handbk. der path. Anat. des Nervensystems, S. 489, 1903.

Occasionally bilateral lesions are found. Secondary degenerations follow softening exactly similar to those following hemorrhage, already described.

The situation of an area of softening due to embolism is not wholly a matter of accident. As the majority of emboli come from the heart it is natural that embolism of the left hemisphere should be more frequent than that of the right hemisphere; (the ratio is five to four cases), the course of the blood current from the aorta being more direct into the left carotid artery. Such an embolus if large enters the middle cerebral artery and stops it, or one of its branches in the Sylvian fissure, thus causing a necrosis in the central area of the brain cortex. If small it is more likely to go directly up with the blood current into one of the branches of the middle cerebral that enters the perforated space. It is found that 60 per cent. of emboli enter these vessels, 25 per cent. enter one of the branches of the Sylvian artery, and the remaining 15 per cent. enter some of the smaller branches of the brain axis. Softening of the cerebellum from embolism is very rare.

The situation of the area of softening due to thrombosis depends on the size and position of the thrombus. It is determined by statistics that thrombi form in the following cerebral vessels in the order named, viz., middle cerebral or its branches, basilar, vertebral, anterior cerebral, and posterior cerebral.

The pathological changes following thrombosis differ in no way from those due to embolism.

The relative frequency of these brain lesions may be gathered from the following statistics. In 1908 consecutive autopsies at the Presbyterian Hospital there were 112 cases of cerebral hemorrhage and 48 cases of cerebral softening. Old scars were found in ten brains, and cysts were found in thirteen brains, all but two in the basal ganglia. In a few cases multiple lesions were found.

The situation of the hemorrhages and softening was as follows:

TABLE IX.—SITUATION OF LESIONS IN APOPLEXY.

	Hemorrhage.	Softening.
Frontal lobe	20	5
Parietal lobe	10	10
Occipital lobe	5	4
Temporal lobe	9	5
Caudate nucleus	6	2
Lenticular nucleus	14	11
Optic thalamus	10	4
Internal capsule	18	6
Lateral ventricle	10	...
Pons	11	1
Cerebellum	4	4
Inferior peduncle of cerebellum	1	...
Under corpora quadrigemina	4	1
On floor of fourth ventricle	2	...
Central part of cerebral hemisphere	1	5
Multiple small hemorrhages	4	...

Etiology. Males are more frequently affected by the vascular diseases than females, and hence death from apoplexy occurs oftener in men. Of one hundred consecutive cases of my own eighty were males. No age is exempt from cerebral hemorrhage. It may occur prior to birth, and the infant may be born with a clot in the brain. Rupture of the vessels is the probable cause of the majority of cases of infantile cerebral palsy which follow traumatism during labor or severe convulsions. Hence the lesion may occur in infancy or childhood. But persons in middle life are most liable, the age from forty to fifty being the age at which the greater number of deaths occur.

TABLE X.—AGE OF ONSET IN APOPLEXY.

	Cases of apoplexy, with autopsy.			Without autopsy.	Total.
Age.	Tooth.	Dana.	Gintrac.	Starr.	
1 to 20	8	4	19	12	43
21 " 30	7	6	19	6	38
31 " 40	17	10	37	15	79
41 " 50	43	11	21	53	128
51 " 60	24	7	19	52	102
61 " 70	20	10	26	41	97
71 " 80	5	5	22	14	46
80 " 90	...	...	...	5	5

When the condition of endarteritis is present any severe effort, physical or mental, any sudden emotional shock, any sudden exposure to cold, a blow on the head, or indulgence in alcohol, or the use of a strong heart stimulant may cause a rupture. Thus, lifting a heavy weight, straining at stool, the effort of coughing violently, or vomiting, and the effort of running fast have been the active causes of an apoplexy in cases of my own. I have seen two cases in children suffering from whooping-cough. I have known a sudden grief and sudden fright, and also a long-continued anxiety followed by disappointment, and in several cases a fit of rage, to be the cause of a hemiplegic or aphasic attack. I have known many cases to follow a debauch. A cold bath has been known to cause an attack, but this I have seen but once, when it was taken soon after a hearty meal. Grant believes that strychnine and quinine in large doses may cause an attack. I have seen two attacks in a young woman subject to purpura. Oppenheim records a case of hemorrhage in the brain following an operation for bleeding piles. The operation was followed by hemorrhage from the lungs and from the bladder before the apoplexy occurred. The sudden arrest of menstruation is said to have caused apoplexy. A hemorrhage sometimes occurs during a severe epileptic fit. This is a common history in children. Cerebral hemorrhage is the cause of death in many cases of chronic nephritis, because of the existence of hypertrophy of the heart and of disease of the arterial walls, which are constant accompaniments of that affection. The increased intra-arterial pressure produced by the force of the blood thrown up from the hypertrophied heart is responsible for the majority of cases of cerebral hemorrhage occurring during Bright's disease. Cases of hemiplegia occurring with or after diphtheria, scarlet fever, and pneumonia are due to rupture of the arteries. It is possible that the toxins of the disease weaken their walls.

The condition of the bloodvessels in purpura may lead to cerebral hemorrhage, also the state of the blood in pernicious anæmia and in leucocythæmia.

The causes of embolism are cardiac disease, with the formations of vegetations upon the valves or of small fibrin deposits on the rough endocardium which are washed into the circulation. In ulcerative endocarditis these contain numerous micro-organisms. Similar emboli may come from the interior of the larger arteries, and atheroma of the aorta is occasionally the cause of cerebral embolism. An embolus in a small vessel may arise from a thrombus in a larger vessel.

Clots may also come from the lungs and even from the veins of the body by way of the larger veins in the lungs. Thus cerebral embolism is a frequent complication of tuberculous disease of the lungs and an occasional complication of pneumonia. I have known two cases to follow phlebitis of the saphenous vein. The exciting cause of the embolism may be a sudden physical effort or a sudden mental or emotional shock.

The cause of thrombosis is any form of endarteritis which narrows the bloodvessel and finally obliterates its lumen, leading to an obstruction of the blood current and to the formation of a clot. Obliterating endarteritis of syphilitic origin is the usual condition leading to such a filling up of the vessel, and as it is a very common condition it is the active cause of the majority of cases of apoplexy in young persons. Senile endarteritis is also very common. The exciting cause of the formation of a thrombus is anything which weakens the action of the heart and thus slows the blood current. Cases of apoplexy occurring during sleep are usually cases of thrombosis.

A general state of weakness, a syncope, the reaction which follows effort, mental excitement, or emotional shock, a fright, or a condition of exhaustion from malnutrition or starvation, may induce such a state of weakness in the heart as to lead to a stasis in the vessels; and if such a stasis occurs in a vessel whose lumen is contracted or whose wall is rough a clot may form. It is not certain whether pathological states of the blood may lead to the formation of clots within the vessels, though this is the theory which accounts for the cases of thrombosis occurring in infectious diseases, in phthisis, in gout, rheumatism, anæmia, chlorosis, leucocythemia, and in the puerperal state.

Symptoms. The symptoms produced by vascular disease in the brain are so nearly alike in cases of hemorrhage and softening that they may be described together. It is admitted that in extreme cases, such as a large hemorrhage or a small embolism they differ widely, and that then a differentiation can be reached. But it has been my experience, from a study of a large number of such cases during life and a comparison of symptoms with pathological findings, that it is not possible to arrive at a positive accurate diagnosis between a clot and a softening in more than one-half of the cases. Hence these symptoms are here discussed together.

Premonitory Symptoms. In all cases of endarteritis the patients suffer from symptoms of disturbance of the circulation in the brain for some months or even years before an actual attack of an apoplectic nature occurs. Miliary aneurisms, however, do not usually produce any symptoms, and may be latent until they burst. The cases which uniformly escape prodromata are cases of cerebral embolism due to heart disease or those not arising from endarteritis.

These symptoms are peculiar sensations of oppression in the head, an internal pressure, the sensation of a band about the forehead, or of fulness and pulsation of the vessels. These come and go irregularly for months, sometimes after physical or, more commonly, after mental effort or emotional excitement; sometimes in connection with atmospheric changes, to which some persons are particularly susceptible; sometimes without known cause. The influence of alcoholic stimulants is more quickly felt by persons who have endarteritis, and in such persons disturbances of digestion are likely to cause vertigo and headache.

Headache is probably the most common symptom of endarteritis. It is usually a dull frontal or occipital pain, rarely unilateral, and varying in intensity from hour to hour while it lasts. It occurs after mental or physical effort or very often after anything which exhausts the strength or depletes the nutrition. It may be due to congestion of the brain, and is then shown by a redness of the face, with distended veins and red eyes and a feeling of fulness and pulsation. It is equally frequent, however, in conditions of cerebral anæmia and malnutrition; for pale, ill-nourished persons, or those who have had exhausting diseases, or who have a weak heart are just as subject to headache as are the plethoric. In such patients, however, there is no congestion of the face and eyes; and the pain is often felt in the vertex. Headache is rarely, if ever, a continuous symptom, and the attacks are sometimes few and infrequent, in other cases very common. Few patients escape prior to an apoplectic attack. The headache in syphilitic cases is usually felt in the afternoon and evening. It is remarkable that very severe and persistent headache often disappears at once after an apoplectic attack. When such an attack is followed by severe or persistent headache the prognosis is most unfavorable.

Attacks of vertigo are among the common symptoms of endarteritis, coming suddenly after efforts, such as straining at stool, running, or lifting weights, or after excitement, or in persons of weak heart, after a meal. They usually alarm the individual, being sometimes, but not often, bad enough to cause staggering or the need of lying down. They are commonly merely of the nature of a sense of swaying, or a sense of uncertain stability, or of a movement of the floor. If a person has a severe attack of vertigo with inability to rise and with attendant faintness and vomiting, it is probably due to the formation of a small thrombus or to a minute hemorrhage in the cerebellum. Such attacks often pass off and leave no permanent trace, but occasionally they are followed soon by more serious symptoms.

Insomnia is a symptom of endarteritis. The patients cannot get to sleep on going to bed, and lie and toss and think and worry; finally they are exhausted and fall into a light sleep in which they twitch and turn and are troubled by distressing dreams, waking in the morning without any sense of rest. This is a condition commonly associated with congestion of the brain. It may be due to too forcible heart action, as in cases of endarteritis associated with nephritis and hypertrophy of the left ventricle, or in cases where the arteries are so rigid as to have lost their elasticity and power of physiological contraction during sleep. After this kind of insomnia rupture of the artery with the formation of a clot is more common than thrombosis. In other cases the obstruction to the circulation causes insomnia by depriving the brain of blood and nutrition. In this condition the patients are unusually drowsy, doze in the daytime, especially after meals, and fall asleep as soon as they go to bed. They sleep heavily, often snoring when this is not their habit, but after three or four hours they wake, and then cannot get to sleep again. The insomnia, therefore, in such patients is worse in the early morning hours. They watch the day break and are early risers. This type of insomnia is more difficult to treat successfully than the other type. For while the former can be cured by warm baths, by hot drinks, and food, by the synthetic hypnotics and bromides, the latter often is intractable. Sometimes a light meal taken in the night on waking, or a heart stimulant, is followed by a nap, but hypnotics are useless. These patients often have their apoplectic attacks during the night, and it is usually due to thrombosis rather than to the rupture of a vessel. When syphilis is the cause of endarteritis the patients usually have difficulty in getting to sleep early ; but they sleep heavily and are wakened with difficulty, being dazed for some moments after waking.

Mental symptoms are frequent prodromata of an apoplexy, and are often present in a state of endarteritis. These may consist of a sense of difficulty in fixing the attention, a defective memory of names, a slight tendency to use one word in place of another, a confusion of thoughts, or even an inability to think without conscious effort and distress, and a marked emotional state due to a lack of self-control. Thus patients may be irritable and impatient on slight cause, may get enraged at little things, or may laugh or cry with hardly any provocation. It is common for these symptoms to be associated with headache and to be of temporary character, passing away with the pain. Patients may suffer for many months from these mental symptoms, and such symptoms may be the only ones observed even in an attack.

Sensations of numbness in one arm or in one leg or in one entire side are not uncommonly complained of by persons who are the subjects of endarteritis, and are very common prodromata of an apoplexy. In two hundred consecutive cases of apoplexy in my private practice of which I have full histories, a complaint of preceding numbness had been made in eighty-six. This consists of a tingling feeling, or the same sensation as when one's foot is asleep. It lasts a few moments or even a day or two, and then passes off. It may be produced experimentally in one-half of the body by compressing the opposite carotid artery, and hence is due to a disturbance of the circulation producing anæmia. When such an attack of numbness wakens a person at night it may cause much alarm. It is not to be confounded with local pressure on the ulnar nerve, as it is diffused over the entire hand; nor with the numb fingers of acro-paræsthesia, which is bilateral ; nor with venous stasis in the limb from pressure which is attended by congestion and pain. It may remain permanently, under which circumstances, an actual rupture of a vessel or a stasis of blood in it has occurred. And after an attack of apoplexy, with or without paralysis, numbness in the affected side is almost always present and remains. Hence, when such attacks of numbness occur they are an infallible warning of the danger of apoplexy. Yet I now have patients under my care who have had many such attacks for several years, but never have had anything more serious. The numbness is rarely attended by an objective anæsthesia. It is a purely subjective symptom. Sometimes a slight awkwardness is associated with it, but when an actual ataxia develops it is because a lesion has occurred.

Ringing in the ears, noises like bells or whistles heard; *flashes of light* before the eyes, or a sudden sense of blindness in one side passing off in a few moments; a subjective sense of a *bad odor* or an *unusual taste* have occurred in different persons as prodromata of apoplexy and as symptoms of endarteritis, but are far less common than the other symptoms mentioned. Nose-bleed is common in persons with endarteritis, and retinal hemorrhages are sometimes found by ophthalmoscopic examination.

The prodromata of an apoplectic attack may occur for many months or years and never be followed by a stroke. They should never be disregarded, however, by patient or physician, and it is better far to take pains to avoid the active causes of a cerebral hemorrhage or thrombosis than to suffer its consequences.

Some form of prodroma had been felt in more than one-half of my two hundred cases.

The apoplectic attack which announces the occurrence of a cerebral hemorrhage, or an embolism, or the formation of a thrombus is always of sudden onset and causes a great mental shock to the patient. Apoplexy, the falling as if struck down, is not always attended by unconsciousness. In fifty-four out of two hundred cases a loss of consciousness occurred. But in the hospital patients observed the proportion was much greater, and in patients who die, unconsciousness at the onset is the rule. The coma is attended by stertorous breathing, by full, slow, tense pulse, not irregular or intermittent, by marked venous congestion of the face, and by complete relaxation of the limbs, so that it is sometimes difficult to determine whether one side of the body is paralyzed. In some cases the head and eyes are turned toward the lesion. The pupils are usually dilated in cases of hemorrhage, the one on the side of the lesion being the larger, and they fail to react to light. In pons lesions they are contracted. The corneal reflex is lost, so that irritation of the eye does not cause a wink. The temperature usually falls at first, and then begins to rise within a few hours, the paralyzed side being a degree higher than the other. There may be an immediate unconscious evacuation of the bladder and rectum, if these are full. If not, there is usually retention of urine during the period of unconsciousness, and the bladder has to be emptied by catheter to avoid its rupture. The state of coma may continue, the temperature may rise to 104° or 105° F., œdema of the lungs and heart failure may follow, and the patient may die within a few hours or on the following day. Occasionally in persons of good vitality the fatal result may be delayed as long as a week, consciousness never returning. In these cases the greater relaxation of the limbs on one side, a manifest drawing of the face and elevation of the corner of the mouth on the non-paralyzed side, with dribbling of saliva on the other side, the failure to elicit reflex movements by pricking one side, the loss of skin reflexes and loss or exaggeration of tendon reflexes, the occurrence of automatic or instinctive movements on the non-paralyzed side, or a marked contracture of the arm and leg, indicate the existence of a hemiplegia. In one girl who never recovered consciousness, and whose lesion was an extensive hemorrhage at the base after fracture, any attempt to uncover the body caused an automatic grasp and effort to draw up the bedclothing on the unparalyzed side. The condition of coma is more likely to occur when the lesion, either a clot or softening, is deep in the hemisphere, is near the lateral ventricle, or involves the optic thalamus. It is also an indication of a large hemorrhage or the plugging of a large vessel. Extensive cortical clots or thrombi almost always cause coma, but small ones do not.

The condition of coma is usually attended by a marked increase of arterial pressure, as shown by the state of the pulse and the sounds of the heart. This is the probable cause of the increased secretion of urine which always follows an attack. The urine if examined is often found to contain albumin and sometimes sugar, which disappear within a week after the attack, and may, therefore, be due to the increased arterial pressure.

The attack of apoplexy may be attended by a convulsion. This may be general, and if repeated is an indication that the clot has burst into the ventricles. It is a most unfavorable prognostic symptom. The convulsion may also be unilateral. This is an almost infallible sign of a lesion of the cortex in the central region.

If the patient recovers consciousness after the attack the mind is usually very dull, and response to questions is for some hours impossible. Then attempts to talk are made, and if there is no aphasia they may be intelligent. Frequently it is some days before the patient recognizes his surroundings. As the mind becomes clearer a definite estimate of the symptoms present becomes possible.

In the majority of cases *paralysis* is found. This was present in one hundred and seventy-seven out of two hundred patients. It occurs in the form of hemiplegia and affects one side of the body only. The face is drawn to the non-paralyzed side, as there is no longer any opposition to the normal tone of the non-paralyzed muscles. This gives an unnatural appearance to the face which sometimes leads the unpractised observer to suppose that the drawn side is the one which is paralyzed. The muscles about the eye which close the lids are not involved, and thus the facial paralysis in hemiplegia differs from that due to an affection of the facial nucleus in the pons or of the facial nerve, in which the eye cannot be closed. The motion of the eyes is not affected except in lesion of the crura or of the corpora quadrigemina. Deviation toward one side may occur during the coma, but this does not persist when consciousness returns. The tongue is also paralyzed, and when protruded turns toward the paralyzed side, being pushed over by the healthy muscle. Swallowing may be difficult and choking frequent for some days after an attack, and this is a sign of unfavorable prognosis. The head can usually be turned freely, though in severe cases the same tendency to turn both eyes and head away from the paralyzed side present in the coma may persist for some days. It is due to the action of normal muscles whose opponents are paralyzed. The arm and leg are completely paralyzed, are relaxed and offer no resistance at first to passive motions. The paralyzed muscles show a normal electrical reaction, and have no tendency to rapid atrophy or to fibrillary twitching.

During the coma and state of shock the tendon reflexes may be lost, but they soon return, and by the end of the second day are exaggerated on the paralyzed side, the knee-jerk being high, and a crossed thigh-jerk being sometimes obtained; that is, a slight adduction of the well thigh and extension of the well leg follows a tap on the patella tendon of the paralyzed side. Ankle clonus is often obtained, and also a retraction of the great toe on irritating the sole of the foot (Babinski's sign). These symptoms continue, even though the paralysis improves. The skin reflexes are lost on the paralyzed side, a sign of great diagnostic importance in distinguishing a true from a false (hysterical) hemiplegia. The body is sometimes paralyzed so that the patient cannot turn in bed, or move his body, or sit up. Inspection of the chest usually shows that respiratory movements are more active on the paralyzed side. The control of the bladder and rectum are lost, and either involuntary evacuations or retention may continue for many days.

As time goes on the hemiplegia may gradually subside. I have known it to pass off in part after three months of complete paralysis, so that the patient eventually walked and signed his name. As a rule, some beginning voluntary motion is evident by the end of two or three weeks, the power of drawing up the leg being the first to return, then to stretch out the leg, then the power to move the ankle, and finally some flexion of the elbow and shoulder. The finer motions of the hand and foot are regained last of all, if they return at all. The face appears straighter in the course of two months. Walking can be resumed after a light attack within a month, but may be regained after a year of paralysis.

Every possible variation in the degree of the original hemiplegia may be observed. In light attacks a sense of weakness only is complained of, and these patients are awkward, but do not lose power in any muscle absolutely, and are soon able to use the hand and to walk. Such patients recover entirely, and may have no trace of the attack at the end of the year. After severe attacks, however, in which the motor tract is injured, and in which a descending degeneration occurs, a partial hemiplegia remains. The muscles gradually become rigid and slow and stiff in motion, the power of finer movements being chiefly impaired; the tendon reflexes are exaggerated, as is also the muscular irritability, so that tapping a muscle causes it to contract.

The gait in hemiplegia is typical. The entire side of the body seems to move together, the body being stiff, the shoulder falling toward the weaker side, the arm being carried in an adducted flexed position with wrist pronated and fingers flexed, the leg being drawn forward with little bending of the knee and ankle, or being carried about in the arc of a circle, the ball of the foot and its inner side scraping the floor. The foot is sometimes turned in and scraped along the floor on its outer side.

When the original lesion has been located in the cortex but one limb may be paralyzed. When it lies in the crus the eye on the side of the lesion cannot be turned inward, upward, or downward; its pupil is dilated, and there is a falling of the upper lid. When it lies in the lower half of the pons a condition of alternating paralysis is produced, the face being paralyzed on the side of the lesion, the arm and leg upon the other side. In this condition the facial palsy involves the upper branch of the facial nerve so that the eye cannot be closed. In a few cases of lesion in the medulla the face has escaped, the tongue has been paralyzed on the side of the lesion, and arm and leg on the opposite side. In hemorrhage into the ventricles, and occasionally in small lesions of the pons and medulla in the raphé, a bilateral or a general paralysis of all the limbs is produced. Thus the original or permanent distribution of the paralysis indicates the position of the lesion. In 200 cases of apoplexy right hemiplegia occurred in 96 cases, being permanent in 73; left hemiplegia occurred in 55, being permanent in 44, alternating paralysis occurred in 4, a lesion of the crus cerebri in 1, and total paralysis in 2. (See page 433.)

In some cases *hemianæsthesia* accompanies the hemiplegia; in a few cases it is the only symptom produced. In the majority of cases of apoplexy it is absent. The symptom is a sensation of numbness in one side, accompanied by a certain degree of loss of the power of perceiving touch, or pain, or sensations of heat and cold. The loss of sensation is never permanently complete in organic lesions. In all cases after the shock has passed away and consciousness is clear some sensation is perceived, especially in the shoulder, or thigh, or foot. One of these senses may be partly lost, the others being preserved. Thus in several of my patients touch was very much impaired, but hot and cold objects and painful pricks were felt keenly. In one case touch was not affected, but pain could not be felt, nor differences of temperature. In two cases the patient had a constant sensation of cold in the left side, could not feel warm objects as such at all, but felt them as cold, and felt touches and painful sensations, but less keenly than on the well side. In one of these cases there was a hemiplegia as well ; in the other cerebellar symptoms without hemiplegia. In another case the sense of pain and of heat was lost, but cold could be felt, and a subjective feeling of cold was present all the time. This patient was slightly hemianæsthetic but had no paralysis. An alternating hemianæsthesia may be caused by lesions in the pons or medulla, as in three of my patients, without any paralysis. The degree of the anæsthesia may vary, being very slight in many patients and only detected by careful comparison of the two sides of the body simultaneously tested. In many cases the hemianæsthesia found immediately after the attack passes off, and in the course of a month sensation is normal. It is only when the sensory tract lying behind the motor tract in the capsule is destroyed, or when the optic thalamus is affected that a permanent blunting of sensation remains. And this is never total, as each hemisphere of the brain receives some impressions from both sides of the body. In 200 cases right hemianæsthesia was found in 21, being permanent in 17; left hemianæsthesia was found in 20, being permanent in 19, alternating anæsthesia was present in 3 and permanent. A loss of tactile memories is sometimes present and indicates a lesion of the cortex in the parietal area. Thus patients who are blindfolded cannot recognize objects placed in the hand. This may occur even when the hand is not anæsthetic. It is termed astereognosis. (See page 438.)

Hemiataxia or an impairment of muscular sense and consequent awkwardness of movement is an infrequent accompaniment of hemiplegia. It may occur alone. It leads to an inco-ordination of motion, both in the gait and in the use of the hand. In 4 cases out of 200 it was noted, and in all it was permanent, in 3 being at first associated with hemiplegia. In one case it was attended by a complete loss of temperature sense, the sense of pain being preserved.

Hemianopsia is a frequent symptom of apoplexy. It is bilateral homonymous hemianopsia, causing blindness in one-half of both eyes. As a rule the patient appreciates the blindness in the eye whose temporal field is blind, and is not aware that the other eye is affected until the physician tests the visual fields. Hemianopsia is usually a permanent symptom. In only 2 out of 13 cases in the series of 200 cases did it pass away. But a gradual increase in the area of the visual field is common, and I have charts which show a great improvement in vision. In the patients who recover the visual tract has been compressed but not destroyed. When the lesion is in the visual tract beneath the cortex the visual fields are more irregular and asymmetrical in outline than when the cortex is affected. Right hemianopsia is more common than left hemianopsia—9 to 4 in my series of cases. (See page 453.)

A loss of visual memories indicates a lesion of the cortex of the occipital lobe and angular gyrus. It is termed mind-blindness or psychical blindness, and when limited to the memories of written language is termed word-blindness. I have seen this as the chief and only permanent local symptom of an apoplexy; the patient, who could talk and write, and was not paralyzed, was unable to recognize friends and familiar places, and was unable to read at all even sentences he had just written. It is usually associated with right hemianopsia. A loss of color vision alone, hemichromatopsia, in one-half of both eyes has been described, but is very rare. I have never seen this condition. (See page 448.)

Aphasia is a common symptom in apoplexy, being associated with right hemiplegia in right-handed people and with left hemiplegia in left-handed people. In two of my cases it was attended by left hemiplegia; both patients were left-handed. It is usually of the form of motor aphasia, but any of the various forms may, of course, be found. Aphasia may occur as the only symptom of an apoplexy; but this is rare. In the 200 cases of apoplexy, 118 patients had aphasia at the onset, and in 84 this condition was never entirely recovered from, though a very marked improvement occurred in the majority of patients when they lived over two months. In 17 of these cases aphasia occurred alone; in the others it was associated with hemiplegia. In 1 case it passed away entirely, though it was the only symptom of the attack, and in another case it subsided wholly after four months. In many cases the aphasia remained after the hemiplegia had entirely passed off. The various forms of aphasia and their significance in determining the location of the lesion have already been considered (page 464).

The *mental symptoms* occurring in apoplexy have not been sufficiently emphasized in text-books. Some cases of apoplexy occur in which they alone are present; confusion of mind, and inability to think, to remember, or to fix the attention, together with a great emotional excitement and lack of control, being felt. In very many cases some mental symptoms are present, and the mind is never as strong, as active, and as useful as before the attack. In some cases these mental symptoms pass away. I have a man under my observation now, perfectly well, who twelve years ago had an attack with loss of consciousness and dementia, with lack of memory lasting a month, and inability to transact business for six months, but with no aphasia or paralysis. In 63 of my 200 cases, marked mental symptoms were present and were permanent. In 2 of these patients delusional insanity finally developed. In 40 a peculiar lack of emotional control was the marked symptom. One man was in a state of constant uncontrollable laughter, would laugh aloud whenever he spoke or made any voluntary movement in his left side, which was partly hemiplegic. In two men frequent crying was complained of, and occurred on any excitement, even when the patients were amused. Such emotional acts may be as uncontrollable as a convulsive spasm would be, and are equally without intention. In several cases such emotional attacks, either of laughing or crying, were common on any excitement. In some cases after the patients recover from the shock of the attack there remains for months a distress on mental effort, a state of bewilderment, in which they lose the thread of conversation, cannot read or write for any length of time without becoming confused, and are forgetful. Some patients lose all recollection of their surroundings, and hence imagine that they are away from home, in a strange place. One such patient, an active intelligent business man, has been reduced to a pitiable condition, without paralysis, aphasia, or dementia, yet so incapable that in reply to almost any question he answers, "I don't know, I can't remember, I am of no use." These patients distrust their own capacity, become vacillating and capricious, are sometimes suspicious, sometimes easily influenced unduly by others. In several of these cases a permanent loss of control of the bladder and rectum has persisted after the attack.[1]

Séguin and Brissaud have noticed that a loss of emotional control occurs more frequently in lesions of the right frontal lobe, and this I can confirm. In many cases in which the mental symptoms are the chief permanent symptoms there has been a slight facial paralysis or a slight hemiplegia or aphasia with the attack, which has subsequently passed off, thus showing that the lesion was anterior to the central or motor region. These patients rarely if ever have hemianæsthesia or hemianopsia.

Cerebellar symptoms, consisting of violent vertigo, double vision, and a staggering gait when the attack is over, may be the chief features of an apoplectic attack. In such cases consciousness is rarely lost, but the patient is usually extremely prostrated, feels weak and greatly alarmed, may vomit, and break into a cold sweat, and remain incapable of moving in bed or of sitting up for some days. All attempts at motion are attended by an increase of the vertigo and a renewal of the nausea and vomiting. The pulse is weak, and may be either very rapid and intermittent or slow. In one such case I have seen Cheyne-Stokes respiration. In my collection of 200 cases there were 4 that presented these symptoms. As patients recover the staggering gait may gradually improve, but there remains a sense of uncertainty and a tendency to fall in one direction. Thus one patient always felt as if about to fall to the right side. She subsequently had a second attack with hemiplegia, and a third attack which left her demented.

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[1] For further cases see the Medical Record, November 13, 1897, "On Some Unusual Forms of Apoplectic Attack."

In severe cases of hemiplegia with hemianæsthesia there is a very marked tendency to the development of *bed-sores* upon the buttock and heel of the paralyzed side. The surface becomes red after slight pressure of the body weight, blebs soon appear, and an ulcerated surface forms which shows a tendency to slough and break down rapidly, so that within a week a deep sore may be present, even exposing the bone and causing necrosis. The lesion in the brain is certainly capable of inducing vasomotor symptoms in the paralyzed side, such as cyanosis and its attendant malnutrition; but it is my conviction that the development of a bed-sore is always due to imperfect care and lack of cleanliness, and that if the patient is turned every half-hour and the skin properly bathed with warm water containing an antiseptic, then with alum-water or alcohol, such sores may be prevented. This is very difficult when the urine and feces are passed unconsciously, and sometimes, with lack of skilful nursing, bed-sores will appear. They are dangerous, as many patients who would otherwise recover die of the septic infection thus produced.

A patient who is paralyzed may have *difficulty in swallowing*, and attempts at feeding may result in food being inhaled into the lungs. Great care has to be taken to prevent this in patients who remain semiconscious for a week or more after an attack. The result will be to develop a pneumonia of septic origin. Charcot believes that the vasomotor paralysis may involve the lung, and in proof cites the frequency with which pneumonia, either septic or spontaneous, develops on the paralyzed side in these cases. Such a pneumonia is a common cause of death after an apoplectic attack, and must be regarded as of serious prognostic import.

Patients who are unconscious during the attack have *retention of urine* and *inability to empty the rectum*. As a rule, the bladder empties itself as soon as it fills up, without their knowledge or control. Sometimes there is a continual dribbling of urine; and for many days after consciousness returns this symptom may continue. It is sometimes necessary to catheterize such patients regularly. It is always well to have a bed urinal in the bed, or pads of absorbent cotton about the genitals, which can be changed frequently. The danger of a septic cystitis is never to be forgotten, as it is the cause of death in a certain number of cases of apoplexy. The rectum may require regular washing out, or, in case of impaction, a digital evacuation.

Irregular Types of Apoplexy. Some very extraordinary forms of attack are occasionally seen. Thus one patient had a sudden attack of vomiting and vertigo, and immediately developed a monospasm in the arm with a right hemiplegia without aphasia. There was no loss of consciousness, but the hemiplegia began with a convulsive motion in the right arm, which continued. Gradually the hemiplegia passed off, but the convulsive movement continued. And for three months the arm was constantly flung about with violent flail-like movements which could not be stopped by the will. These were more violent than athetoid motions, and were still continuing at the last report.

In another patient I saw a series of convulsive attacks, five occurring in the course of seven days, each limited to the left side of the face and arm, each lasting a quarter of an hour, and each causing a temporary paralysis which by the end of three weeks had entirely passed off. The only permanent symptom was a slight degree of mental dulness and apathy with incapacity for business.

In another patient a series of attacks of a convulsive nature limited to the right leg occurred during ten days; each attack was very violent, so that the entire body was shaken, and lasted for nearly half an hour, leaving the leg paralyzed. Then the attacks ceased, the condition of paralysis gradually subsided in six months and she has had no return during the past seven years.

One man suffered from a sudden distress in his head, followed in an hour by a general convulsion, which left him with right hemiplegia and aphasia. During the following three months the condition of paralysis entirely subsided and he recovered his speech, but never recovered his mathematical faculty. He cannot do the simplest sum. This first attack occurred in 1887. Since that time until the present (1903) he has had, every six or twelve months, sudden attacks consisting of confusion of mind, complete inability to speak, though he understands what is said, a twitching which begins in his fingers and thumb, and gradually involves the whole right arm, then the right side of the face, and then the right leg, attended by a tingling of the entire right side. This convulsion lasts several hours, then ceases slowly and leaves a paralysis which passes off entirely during the following two days. I know of over twenty such attacks having occurred during the past fifteen years. In the interval he appears hearty and vigorous, though now eighty years old, and he walks five miles a day. He has some difficulty in talking, finds words slowly, but is not aphasic. His memory is poor. He has no cardiac disease, but his arteries are tortuous and hard. I have similar histories in three other cases. In all these cases the probable lesion was cortical hemorrhage, or thrombosis, as spasm is considered a sign of cortical irritation.

General convulsions do not often occur in an apoplectic attack in adults unless the clot bursts into the lateral ventricle. In children, however, they are very common at the onset, and, as we shall see in discussing infantile hemiplegia, convulsions come either with or after the attack in the majority of the cases. In two young girls, patients of my own, where the hemiplegia occurred during an attack of whooping-cough, general convulsions accompanied the attack. In adults their occurrence is of bad import, but in children this is not so.

In one patient who had a sudden attack of left hemiplegia at the age of sixty-eight years, a violent tremor developed in the left hand soon after the attack. This persisted for five months until his death The hand and arm soon became cyanotic and œdematous, were covered with cold sweat, and were wholly paralyzed. He became violently insane soon after his attack, had well-marked hallucinations and delusions, did not sleep for two months, and finally died of exhaustion.

In several patients a febrile movement has persisted after the apoplectic attack, and severe headache with insomnia of an obstinate kind, not much relieved by anything excepting large doses of morphine, have appeared. In these patients the hemiplegia has shown no tendency to improve, and in all a fatal result has followed within two months. I look upon this combination of symptoms with much dread, and consider that in all such cases an acute inflammation must be set up in the brain about the clot or softened area.

In the cases where the clot invades the ventricles general convulsions are usual, are frequently repeated, the paralysis affects both sides of the body, sometimes a rigidity of the limbs follows, the pupils are widely dilated, the temperature falls, the pulse is slow, and respiration is labored and stertorous. Such an attack may follow a milder attack after twenty-four or forty-eight hours, the original clot breaking through into the ventricle. Hence the need of keeping patients quiet after even a slight attack. These patients usually remain comatose and die within twenty-four hours, of cyanosis and asphyxia. Yet in one hospital case I have seen a gradual recovery after two weeks of unconsciousness attended by all these symptoms, and the final result was an extreme state of hemiplegia which remained six years to my knowledge without recurrence. But one patient in my series of 200 cases showed symptoms of ventricular hemorrhage, and died in the attack.

Bulbar paralysis of the apoplectic type is illustrated by the following case: A man previously healthy but alcoholic was suddenly attacked at the age of fifty-three years with weakness of the entire left side, followed in a few minutes by paralysis in the entire right side and attended by difficulty in speech and in swallowing. There was no headache, no convulsion, and no loss of consciousness. His condition had been stationary for five years, when I saw him. His eyes were normal. There was no weakness of his face, but his speech was thick, voice loud and husky, and not under control, and swallowing and articulation were very imperfect. His tongue protruded, but was thin and had a marked tremor. He was very emotional, laughed and cried without cause. His power in arms and legs was good, but he had an extreme degree of ataxia in all motions of hands and legs, a very ataxic gait, increased by closing his eyes, but his knee-jerks were normal. His control of his bladder was imperfect. He had no pain, no vertigo, no deafness. The probable lesion was a small hemorrhage in the raphé of the medulla affecting both interolivary tracts, and his ninth and twelfth nerves. Such cases have been observed and the lesion found by Senator, Meyer, and Spitzka.

Attacks of Apoplectic Naturé without Loss of Consciousness. In the majority of cases of apoplexy a loss of consciousness does not occur. Under these circumstances, at the time of the attack the patient feels suddenly a sense of pressure in the head, or a dizziness, with headache, or finds it necessary to sit down, or is able to drag himself slowly to a chair, or falls if he cannot reach a seat. He then notices a numbness in one limb which extends to the entire side of the body, and is attended by some weakness in the arm and leg. Or he becomes confused in his mind and cannot find words, and soon develops aphasia. Or his numbness increases, his arm becomes ataxic, and he moves his leg with difficulty. Or he suddenly becomes conscious of a blindness in one eye which on examination is found to be a hemianopsia. Or he feels bewildered and confused, and can no longer understand what is said to him. Or he is dizzy and staggers in walking. Anyone or any combination of these symptoms may come on suddenly, may recede, and then return, to leave the patient finally in a state of incapacity of some kind. As a rule, when the attack is slight and not accompanied by unconsciousness or followed by fever, there is a fairly rapid improvement; and after two weeks the patient is able to sit up and begins to recover his lost power. But a complete recovery does not often follow, and some trace of the original symptoms usually remains. Aphasia is not wholly recovered from, some hesitancy of speech or tendency to misplace words remains, some local symptoms continue, and a trace of hemiplegia is seen in his gait for the rest of his life. If the symptoms pass off entirely, as they did in 16 of my 200 cases, the patient may remain well for some years, but the probability is that a second attack will occur.

Attacks of Gradual Onset. Thus far attacks of a sudden nature have been considered. But in a certain number of cases the onset of the nervous symptoms is gradual. Thus in several of my patients four or five days have elapsed before all the symptoms have appeared. In these cases the mental dulness and apprehension, the sense of something occurring in the head, with pain, vertigo and weakness, are present throughout the period of onset. In some cases aphasia first appears, then becomes more complete; right hemiplegia is added, and finally hemianæsthesia. In other cases the paralysis advances slowly from face to arm, and then to leg, or in the reverse order, and if right sided, aphasia may finally develop. In some cases hemianæsthesia precedes the hemiplegia. In some hemianopsia first occurs, and then a hemianæsthesia or sensory aphasia, which may subsequently subside. In still other cases the mental symptoms become more and more marked, confusion of thought is evident in speech and action, and a dementia finally develops, often attended by emotional excitement, crying or laughing without cause. In some cases monoplegia, or monospasms, or a loss of sensation in one limb only, is the final result, the initial symptom being more extensive than the final ones. It is in these cases of slow onset that the various types of aphasia are particularly frequent, and psychical blindness or deafness or some form of subcortical aphasia persists.

The *recurrence* of apoplectic attacks is a danger to which all patients are liable. Twenty-two patients among my two hundred cases suffered from more than one attack, one died in his fourth and one in his fifth attack. I have known one person to survive seven attacks, and many to survive the third which, according to popular superstition, is uniformly fatal. The same person may have a recurrence of the same symptoms in each attack, or may have different symptoms each time, depending wholly upon the bloodvessel which is affected. One man, aged seventy years, had an extraordinary series of attacks. The first and second were attacks of right hemiplegia with aphasia, the third was of left hemiplegia. These occurred on successive days and lasted a few hours. Five days later he had a fourth attack. During the following ten days he had three more, one right-sided, two left-sided. All this time he was growing more feeble, but after each attack the paralysis passed off in a few hours. Six days after the seventh attack he had an eighth which left a permanent left hemiplegia from which he died four days later. His arteries were rigid, his heart was weak, but the sounds were clear. The diagnosis made was multiple thrombosis, and he was treated by nitroglycerin and strychnine, the administration of which in each attack seemed to be followed by a recovery from the paralysis. The autopsy showed multiple areas of softening due to thrombosis. In several cases multiple lesions were found at autopsy.

There is no rule to determine the interval between attacks. A few months, a year, or even ten years may elapse before the recurrence. The average in twenty-two cases showed that the chance is in favor of a second attack within two years of the first one. But one of my patients did not have his second attack until fifteen years after the first one.

The Terminal Condition. The course of the disease after an attack of apoplexy is usually one of slow improvement and a greater or less degree of recovery. The terminal condition may be much more favorable than the original symptoms would lead one to suppose.

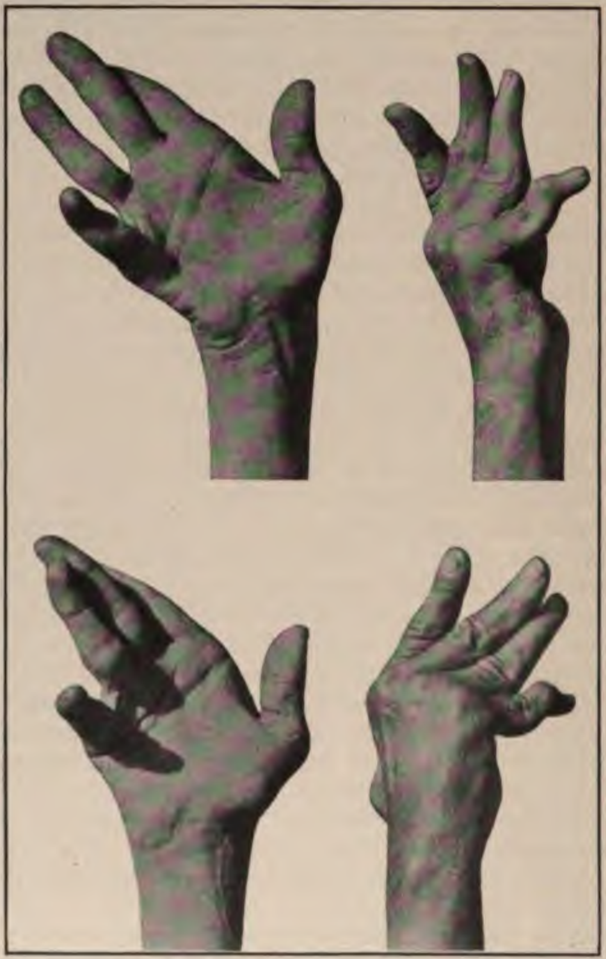
Many patients remain in a condition of partial mental enfeeblement with less control of the emotions than before the attack, and some defects of memory, especially of events occurring about the time of and subsequent to the attack.

Many patients suffer from a partial hemiplegia. The face recovers almost entirely, a slight flatness on one side, and a little deviation of the tongue only remaining. Often the facial expression of amusement or of grief is perfectly shown when voluntary movement is at fault. Sometimes, as in one of my cases, the voluntary power returns in the face, but the paralysis is evident when emotion is shown. This patient had hemiplegia with hemianæsthesia. The arm is usually permanently paralyzed to a greater degree than the leg, and is more commonly subject to contractures. The flexor muscles are usually contracted and hold the elbow flexed, the wrist flexed and pronated and the fingers closed, and the extensors and supinators being weak cannot overcome them. In one patient the arm, forearm, and fingers were rigidly extended, the fixed position being similar to that assumed in reaching out for an object a foot away from the hip. The consequence is a marked deformity of the hand in severe cases and an awkward movement in light cases. A rigidity of the muscles develops within two months of the onset, or sooner, so that even passive motion is difficult. This may sometimes be overcome for the time being by massage, by rapid motions of flexion and extension of a limb by hot applications, or by an Esmarch bandage. As a rule, the rigidity is absent when the patient is asleep, and only returns after waking when he begins to move the limb voluntarily. The limb may be so stiff as to be painful, and pain on motion of the joints is a frequent complaint in hemiplegic patients. The arm is usually held close to the side, and complete abduction or circumduction at the shoulder is rarely possible. The leg is also rigid, is moved as a whole, and is held straight. The flexors of the knee and the extensors of the foot are paralyzed to a greater degree than other muscles, and their opponents are held rigid. Hence the knee is not bent in walking, and the foot often assumes an equino-varus position. In very severe old cases in which the patient is confined to bed, an extreme flexion of hip and knee with adduction of the thigh may develop. The rigid muscles may be hypersensitive to percussion or may be too rigid to contract when tapped.

The tendon reflexes are uniformly exaggerated on the paralyzed side, and not infrequently on the healthy side as well to a less degree. Ankle clonus is easily obtained, and in extreme cases of hemiplegia I have obtained a clonus by depressing the patella, by depressing the toes, by extending the wrist or fingers or thumb. In cases which recover entirely from paralysis this increase of tendon reflex may remain. Babinski's reflex is always present.

In some cases subsequent to a hemiplegia a slow spontaneous but involuntary movement begins in the upper extremity, producing flexion and extension of the fingers and hand and often extensive motions of the elbow and shoulder. These are more rarely seen in the foot and in the face. This is athetosis. It is increased by any voluntary effort of the paralyzed or of the healthy hand, and cannot be arrested. Emotional excitement also increases the motion.

FIG. 213.



Athetoid movements of the fingers. (Curschmann.)

This symptom is seen in infantile hemiplegia oftener than in adult cases, but it occurs in adults. I have known it to cause peculiar grimaces in the face and an interruption of speech from contortion of the muscles of the face and tongue. Hammond ascribed it to a lesion of the optic thalamus, but it is now regarded as an evidence of cortical irritation. When such symptoms are due to lesions in the optic thalamus it is probable that the lesion causes an irritation of sensory fibres which is conducted to the cortex, and there gives rise to motor impulses. Thus in several children suffering from athetosis no cortical lesion has been found at operation. The most pronounced case in an adult that I have seen developed after an operation for the removal of a brain tumor from the posterior central convolution, and continued for a year after the operation, subsiding slowly as voluntary power was regained. I ascribed it to pressure from a clot which was gradually absorbed. Hemiathetosis is, therefore, an occasional sequel of an apoplectic attack. Hemitremor is also occasionally developed, a tremor which may be constant even during rest, or which may only occur on voluntary motion. It occurred in but one of my cases.

The lack of voluntary exercise usually leads to a slight atrophy of the paralyzed muscles. An extreme and rapidly advancing condition of atrophy without any change in electrical reaction has been occasionally observed, and cannot be explained.

The joints occasionally become painful, probably from disuse, and require massage and passive motion. A true synovitis with hemorrhage into the joint sometimes develops, as Charcot pointed out, but this is exceedingly rare. I have never seen a case, and question any causal relation between the apoplexy and its occurrence.

A condition of hemianæsthesia, hemiataxia, and hemianopsia occasionally remains. The first brings with it a disagreeable sense of tingling or numbness, and leads to awkward movements even when ataxia is absent. The second leads to marked inco-ordination, of finer motions, and patients drop objects from their hands when not looking at them. Hemianopsia is rarely recovered from, but patients become accustomed to having but one-half of the field of vision clear, and guard against accidents from objects approaching the blind side. Deafness from brain lesion is so rare as to be a curiosity. Mills has reported a case with bilateral destruction of the temporal lobes. These defects of sensation are much more likely to remain stationary than is the condition of paralysis. Any one of the many forms of aphasia already described may persist after a vascular lesion. In fact it is largely from the study of small local areas of softening in the brain due to embolism or thrombosis that our knowledge of the possible forms of aphasia and apraxia is due. It is not necessary to recount these symptoms here. The peculiar combinations of cranial nerve symptoms with hemiplegia characteristic of pons and medulla lesions need not be described, as they have been considered in the chapter on diagnosis of local lesions.

Diagnosis. An apoplectic attack may be distinguished from an attack of syncope by the pallor, stoppage of heart, and weak respiration, dilatation of the pupils, preservation of knee-jerks, rapid recovery, and absence of coma, or subsequent local symptoms of a brain lesion in syncope.

An epileptic attack, with cry, biting of the tongue and general convulsion is followed by a state of coma which may be mistaken for apoplexy when no history of the onset can be obtained. But an epileptic's tongue is scarred, and there are usually scars upon his head or extremities as evidence of prior attacks. Epilepsy is a disease of youth, and the epileptic rarely shows signs of the vascular conditions which lead to apoplexy. The coma is not very deep, the patient when aroused shows no paralysis, the pupils are equally dilated, there are often hemorrhages in the conjunctiva and capillary hemorrhages on the face. And within an hour or two returning consciousness enables a history of prior attacks and the absence of any paralysis to be established.

The coma of alcoholic intoxication is not as deep as that of apoplexy, and the drunken man can usually be aroused, and gives evidence of his condition by his breath, his dirty appearance, his maudlin resistance, his delirium and restlessness, and his lack of paralysis. The corneal and pupil and tendon reflexes are preserved. It should not be forgotten, however, that some cases of apoplexy occur in a state of intoxication, or from traumatism in this state, and symptoms of compression of the brain, unilateral paralysis, or inequality of the pupils, should lead to careful watching in any case of doubt. The signs of alcoholic intoxication will pass off in a few hours, a brisk purgative or a large salt enema hastening recovery.

The coma of opium poisoning is attended by very slow respiration, small, rapid pulse, cool skin of blue color, and extreme contraction of the pupils; it is never as deep as the coma of apoplexy, and never attended by unilateral symptoms or loss of the corneal or tendon reflexes.

A uræmic condition leading to convulsions and coma may be mistaken for apoplexy if a full history of the preceding symptoms of nephritis is not obtainable. But the symptoms of œdema of the tibiæ and face, the urinary odor of the breath, the small amount of bloody urine of high specific gravity and loaded with albumin and containing casts will point to the correct diagnosis in cases of acute Bright's disease. Uræmic coma is the culmination of an attack which begins with vomiting, headache, and convulsions. In cases of chronic cirrhotic kidneys apoplexy is a common occurrence, and hence in such cases the finding of casts and albumin in small amount, with urine of a low specific gravity, does not aid in the diagnosis. And here it is only by the absence of the active local symptoms of apoplexy, especially the lack of unilateral paralysis or difference in reflex actions on the two sides, that a diagnosis is to be reached. It is often wise to watch a case for twenty-four hours before arriving at a conclusion.

The same may be said of diabetic coma, though here the examination of the urine is of far greater value, as the glycosuria which follows a cerebral hemorrhage is rarely as extreme as that found in diabetes. The odor of acetone is unmistakable, and is not present in apoplexy unless this is associated with diabetes. In cases of doubt reliance must be placed upon the previous history, as diabetic coma is not of sudden onset, is usually preceded by headache, anxiety, distress, and sensations of suffocation, followed by somnolence. But it is to be remembered that diabetic patients sometimes have apoplectic attacks.

An apoplectic attack may be the first symptom of general paresis, or may occur at any time in the course of the disease. The symptom will then be due to a hemorrhage in the meninges, usually a dural hematoma, and will be identical with those of a cortical cerebral hemorrhage at the time of the attack. The history of mental symptoms of a grandiose kind, of mental failure, and loss of memory; of attacks of emotional excitement; of disturbances of speech; of unusual physical efforts in a person really weak; of exaggerated knee-jerks, and other signs of paresis, will lead to the diagnosis. And the result will be a much more rapid and complete recovery from the hemiplegia or aphasia produced by the attack than is ever seen in a hemorrhage which destroys the brain.

The differentiation between cerebral hemorrhage, thrombosis, and embolism is always difficult and often impossible; but when the characteristic symptoms of any one of these affections are present a probable diagnosis can be reached.

In **hemorrhage**, if the person is young, there is a history of whooping-cough, of convulsions, or of purpura; if old (and the majority of cases occur after the age of forty), the individual is ruddy, muscular, or corpulent, has a history of nephritis or of endarteritis, has no history of syphilis, has no cardiac murmur, but has a strong and possibly hypertrophied heart. There is usually a history of mental excitement or physical exertion preceding the attack, but rarely any history of premonitory vertigo or mental confusion. There is a history of very sudden loss of consciousness, with flushing or cyanosis of the face, a marked pulsation of the bloodvessels, the pulse being full, slow, and irregular, a slow stertorous respiration, and deep coma from which these patients cannot be aroused. There is often a difference in the size of the pupils, and they fail to react to light, and the eyes are turned to one side. During the first day there is a fall of temperature to 97.5° F., followed by a slow rise to 102° F. A rigidity of the paralyzed limbs occasionally appears, but unilateral twitching or convulsions are rare. The paralyzed side at first feels cooler, but when the temperature of the body begins to rise it is often found to be a degree higher than the other side. The paralyzed extremities are sometimes slightly œdematous, often cyanotic, and sweat freely. The urine often contains albumin and sugar after a hemorrhage. When the patient recovers consciousness after twenty-four hours, or not until after two or three days, he is much dazed, comes to himself very slowly, complains of pain in the head, is restless, and tries to move; is sleepless, but is very dull mentally and incapable of showing an appreciation of his condition for at least a week. Hemiplegia is almost always present; often associated with hemianæsthesia, and sometimes with hemianopsia, though the two latter symptoms may subside within the first ten days. Aphasia is almost always present if the right side of the body is paralyzed, but is rarely one of the pure special types, such as word-deafness, or word-blindness, or a pure motor aphasia. Bed-sores occur more frequently in cases of hemorrhage than in thrombosis or embolism, and so does pneumonia. During the month following a hemorrhage there is a gradual but steady improvement in both mental capacity and in the local symptoms. Some permanent defect, however, is always left with signs of secondary degeneration in the motor tract in the majority of cases. Retinal hemorrhages occasionally may be seen with the ophthalmoscope.

In **thrombosis** the patient if young has a history or physical signs of syphilis, and if old is the subject of endarteritis, his temporal arteries being tortuous, his radial arteries hard, his pulse irregular, and his second heart sound accentuated. There may be a history of some acute illness, especially of an infectious type, just prior to the attack. There is a history of some premonitory symptoms extending back several months, such as headache, vertigo, or insomnia, even if there have been no slight attacks of numbness or of paralysis. It is particularly in these cases that the prodromata which have been described occur. There is no nephritis, and the urine is normal. There may be a history of some shock or fright, of some general sense of faintness or weakness just prior to the attack. There is usually a slow onset of the attack, giving a few moments of alarm and conscious illness; in fact, the patient may feel his paralysis coming on before consciousness is lost. In the majority of the cases there is no loss of consciousness. The face is pale, the pulse normal, not full or slow, the respiration is regular, and there is no fall of temperature. The pupils are equal and react to light. The coma is not very deep when it occurs, and irritation of the body produces automatic movements which show one side to be paralyzed. Twitching of the limbs is common, convulsions rare. The coma does not last more than twelve to twenty-four hours, and when consciousness returns the mind is clear, at first, though excitement and even delirium often follow. There are no signs of increased intracranial pressure. The local symptoms are less extensive than in hemorrhage, and rather more so than in embolism. They show considerable variation in the first week. Thus a hemiplegia may pass into a monoplegia, a hemianæsthesia may wholly disappear, and a general aphasia subside into some special type, as word-deafness or word-blindness. In other words, the compensatory collateral circulation in the brain being established and the initial œdema subsiding, the functions at first suspended may be resumed. But the area of the brain permanently softened does not get back its power, and hence permanent localizable symptoms of small extent remain, and these show little tendency to improve. Mental symptoms of the nature of impairment of memory, loss of emotional control and excitement as permanent symptoms are more common in thrombosis than in hemorrhage or embolism, and both precede and follow the attack. And there is a marked tendency to recurrence of attacks, usually without loss of consciousness, with a renewal of symptoms which have in part subsided, or with new symptoms of a local kind. The ophthalmoscope may show arterial sclerosis in the retina.

In **embolism** the patient is usually young and has an audible heart murmur or a history of phlebitis, endocarditis, rheumatism, or infectious disease; is not the subject of nephritis, and has no signs of endarteritis. He has no special apoplectic appearance, and is pale rather than ruddy during the attack. The attack occurs without premonition, and not after effort or motion. It is not always attended by a loss of consciousness. When coma occurs, and as a rule it does not, it is not very deep, and irritation of the body usually causes some automatic motions, even if there is no semiconscious response. The pulse is not full and not slow, but is such as would be present in the cardiac state found. The respiration is rarely stertorous, and the temperature does not fall. The pupils are not unequal in size and usually react to light. Unilateral spasms, or twitching of the paralyzed limbs, or convulsions beginning in one limb and extending to others, finally becoming general, are not infrequent, and during the attack and after consciousness is regained these may recur. There is no difference in the temperature of the two sides, and no œdema or cyanosis. When the patient recovers consciousness he does so rapidly, does not complain of symptoms of pressure in the brain, but often passes into a state of delirium which may last some days. Extensive local symptoms are much less common than limited ones. Thus some special type of aphasia easily distinguished from other types, monoplegia rather than hemiplegia, or hemianopsia alone are the symptoms found after the third day. There is often a very surprising improvement in all the local symptoms on the second day, with a return of them on the third or fourth day, followed then by a very slow improvement. Sometimes the local symptoms are permanent. In other cases they may entirely pass away, and no trace of the attack may remain.

The ophthalmoscope may show embolism of the central artery of the retina.

When in any case the symptoms mentioned are present there is little doubt as to the diagnosis. But in many cases there is an absence of many of the distinguishing symptoms, and a differentiation is impossible. This fact is substantiated also by autopsy records. Thus in twenty cases of supposed embolism where cardiac disease was present and the attack was fairly typical, nine were found after death to have been cases of hemorrhage (Kleber[1]). It has been my experience that in few conditions are errors of diagnosis more common than in apoplexy.

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[1] von Monakow. Gehirnpathologie; Spec. Path. u. Ther. von Nothnagel, Bd. xi. S. 776.

Prognosis. The prognosis in an apoplectic attack depends somewhat upon the cause of the apoplexy. Cerebral hemorrhage is much more likely to be fatal than thrombosis or embolism. It also depends upon the underlying cause of the attack and the prognosis of that affection. The first attack is less likely to be fatal than subsequent attacks. The older the patient the less likelihood of his recovery. Very deep coma, its duration for more than a day, a rapid fall of temperature, or a rapid rise of temperature, the development of Cheyne-Stokes respiration, heavy stertor, the deviation of head and eyes to one side, are all important and very unfavorable symptoms. If convulsions occur with signs of increased intracranial pressure a ventricular hemorrhage has probably occurred and death is sure. Retinal hemorrhages are of bad import, as they show a high degree of pressure in the skull, and hence a large clot. Bilateral paralysis, especially if attended by cranial nerve palsies, shows a lesion of the pons or medulla which is almost surely fatal. It is not to be forgotten that a slight attack may be followed immediately by another, more severe, and this by a fatal attack. If the patient recovers consciousness after the attack the development of delirium, of high temperature, of insomnia, of great restlessness, of pain in the paralyzed limbs, a tendency to bed-sores, and the failure to control the sphincters make a fatal termination within two weeks probable. If few or none of these symptoms occur, and the temperature becomes normal within a week, and some slight improvement in the local symptoms is manifest, life will probably be spared.

The greatest caution must be exercised in giving a prognosis on the degree of recovery from the local symptoms.

Aphasia which is general at the outset always passes away in part, and has, in my experience, more chance of improvement than any other local symptom. A pure type of aphasia, motor or sensory at the outset, is usually permanent, though careful education enables a patient in the course of time to regain his speech at least to some degree. Aphasia alone without hemiplegia may be entirely recovered from, or may remain permanent and incurable. In 56 cases of attacks of aphasia 14 recovered the use of speech completely.

Hemiplegia, even if total, may not remain. It usually passes off in part, and it is safe to predict that the patient will walk again, even in severe cases. In one case under my care the patient walked well at the end of a year, though he had made no voluntary movement whatever in arm or leg at the end of three months. In another case a year elapsed before the patient could stand alone, yet two years later he walked without a cane. The arm and hand are always more completely and permanently paralyzed than the leg, and many patients who can walk well never regain power of writing or of feeding themselves. Yet I have seen complete return of all but the very finest of motions, such as the power to pick up a pin or to button the clothing. The increase of reflex in the knee and ankle is not an unfavorable sign. But if contracture of the limbs develops after the first month a complete recovery never takes place. In the series of 200 cases here collected, of 177 patients with hemiplegia 22 recovered completely, and in every patient who lived beyond the second month a very marked improvement occurred. The sooner the return of any motion the more complete will be the eventual recovery. The paralysis commonly passes away from the face entirely, so that only on very careful testing is a difference in the two sides to be seen. This is probably because even in bad cases the instinctive movements of facial expression are not affected, excepting when the optic thalamus is destroyed, and the control of the face is bilateral in many persons. The tongue usually recovers with the face. It is evident, therefore, that a promise of much improvement in the power of motion may almost always be given. Post-hemiplegic athetosis and tremor rarely if ever subside or improve.

The prognosis in regard to sensory disturbances is less favorable than in regard to paralysis. The initial hemianæsthesia usually subsides. But when it persists more than a month it never wholly leaves the patient, and it is usually attended by a feeling of tingling and numbness which gives rise to much discomfort. This numbness also makes a patient awkward in his movements, even when it is not associated with paralysis.

Hemianopsia when due to shock or to pressure on the visual tract usually passes off within ten days or two weeks. If it persists beyond that time it is due to a lesion of the tract or cortex, and is incurable. I have never seen the symptom of hemianopsia pass away when it had persisted one month. The limits of the visual field may extend a little and the patient may learn in time to disregard and even to forget his blindness, but a perimetric examination usually shows that it is stationary.

The irregular motor and sensory and cranial nerve symptoms due to lesions of the crura, pons, and medulla, when they are not followed soon by a fatal ending are usually permanent and have a bad prognosis. They are due to a rupture of small tracts which never unite, and are followed by secondary degenerations which are permanent.

The cerebellar symptoms often pass away, but a tendency to vertigo may persist.

The mental symptoms attendant upon an apoplexy are never absolutely recovered from, and after each attack they are more marked and permanent. When they are due to the underlying endarteritis it is to be expected that they will be even worse after than before the attack. The actual obscuration of ideas following the attack, the inability to comprehend, to think, to judge, and to act slowly subside. The flow of ideas becomes clearer, and if there is no aphasia it may be possible for a man to make a will within two weeks of the attack. But subsequently to an attack the mind may be permanently enfeebled, and when an emotional state develops, and the memory is easily confused, and the effort of continuous talking or reading causes distress for two months after an attack, it is a question whether it will ever be possible for the man to resume his occupation. Yet it is often amazing to see how accurately a business man can judge of his affairs, and how clearly, even though aphasic, he can indicate his desires in regard to important matters while severely paralyzed. In the series of 200 cases, mental symptoms were permanent in 63. But in several cases where more than two slight attacks occurred it was these symptoms which remained and which rendered the patient incompetent after the aphasia and hemiplegia had entirely subsided.

A very rapid improvement in all the local symptoms after an apoplectic attack should not invariably be regarded with favor, as this is the history in attacks occurring in cases of general paresis. Such a recovery should awaken a suspicion of this affection, which, if found, will greatly change the prognosis.

It is always to be remembered that, excepting in children, one attack of apoplexy predisposes to another. Endarteritis is a steadily progressive disease. No prognosis can safely be given as to the length of the interval between attacks, but a second attack is reasonably sure.

The prognosis in cases due to syphilitic disease of the arteries is not more favorable than in other cases; for no amount of antisyphilitic treatment can affect the condition of softening due to a thrombus in an artery the subject of obliterating endarteritis. It is, therefore, a mistake to give a good prognosis in syphilitic cases or to promise any more from treatment in such cases than in others. In fact these patients are more liable to recurrence than others, and hence the prognosis is worse. An obliterating endarteritis is not to be cured. My experience in this respect is quite the reverse of that of von Monakow.[1] The eventual prognosis after the end of the sixth month is much worse in cases of embolism and thrombosis than in hemorrhage. For in the former the symptoms then present show the permanent effect of softening, while in the latter a shrinkage of the clot and its scar or cyst may go on for two years.

Treatment. For a person who has beginning endarteritis it is essential to adopt a manner of life which will arrest the progress of the affection and prevent its most serious result. It is generally agreed that a diet which is simple in character, without rich sauces and strong condiments, and which does not contain an excess of nitrogenous matter, is the one least likely to increase the arterial disease. Patients therefore should not eat a great deal of meat, eggs, or cheese, and many are better for avoiding milk. It is most important to avoid alcohol in any form, and the use of wine, beer, and spirits should be forbidden. Strong coffee and tobacco are also supposed to induce endarteritis, and should be used in moderation only. A proper care of the digestion is therefore imperative. An annual visit to Carlsbad for a mild course of treatment, or to Saratoga for a corresponding cure, is most advantageous. The habitual use of water both with and between meals is to be urged. A fair amount of exercise not of an exhausting character is to be commended, golf and horseback riding being advisable as equally good means of out-of-door exercise, and billiards or Indian-club swinging with light clubs the best indoor forms. Tennis, or lifting weights, or rowing are to be avoided, as they put too severe a strain upon the heart for a person with endarteritis. Exercise should cause a mild perspiration, but if followed by exhaustion or palpitation, is objectionable. Many persons live more comfortably without exercise, but if the lack of it induces obesity it must be taken. Tepid or cool baths are preferable to very hot ones, and the sudden shock of cold is to be avoided. A climate should be sought without great extremes of heat or of cold, and chills should be guarded against. As the accurate observations of Carter[1] have proven that iodide of potassium does not decrease pulse tension, I see no reason to believe that its continued use in small doses, which is advised by many authors, can in any way prevent arterial disease. But a person who has had syphilis should certainly take a course of mercury every three years and a course of iodide of potassium every six months, to prevent the development of endarteritis.

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[1] Gehirnpathologie; Spec. Path. u. Ther. von Nothnagel, Bd. ix. S. 870.

Patients who have any arterial disease should avoid sudden exertion, and this is a good rule for everyone over forty-five years to follow, as many cases of apoplexy occur after exertion in apparently healthy persons. The existence of miliary aneurisms is often unsuspected until they rupture after a sudden effort. Thus one of my friends, a healthy woman of fifty years, lifted a trunk and immediately had a cerebral hemorrhage which killed her. It is just as imperative in persons with endarteritis to avoid exhausting effort as it is for one who has heart disease. And mental or emotional excitement of an intense kind is equally to be avoided. Contentment, with a moderate degree of success and wealth, is better than fame or riches bought by an apoplexy. These precautions should be particularly urged upon patients who are known to have nephritis.

The treatment of an attack of apoplexy depends upon the cause, as hemorrhage, or softening, require exactly opposite measures.

If it is apparent that a hemorrhage has occurred the patient should not be carried far, or jolted in the process, but should be placed in bed with his head somewhat higher than usual, and should lie upon the non-paralyzed side rather than upon the other, or upon his back. Respiration is always hampered by the accumulation of saliva, by a falling backward of the larynx, and by paralysis of the respiratory muscles on one side. If the patient lies on the non-paralyzed side the saliva runs out, the larynx does not become obstructed, and the weak side expands better than if it is held down by the weight of the body.[1] If gravitation acts at all on the clot, it should not favor its rupture into the ventricle, hence the side of the brain which is injured should be against the pillow. The full, beating pulse which tends to increase the bleeding is often the result of carbonic acid poisoning from imperfect respiration. All obstructions to breathing, tight clothing or neck-bands should, of course, be removed. The mouth should be wiped out frequently with an alkaline antiseptic solution. All exertion, such as swallowing, and the consequent coughing which inevitable choking produces, are to be avoided, hence no attempts at medication by the mouth are to be tried during the coma. Vomiting is always dangerous to the patient, from the strain and the possibility of inhaling particles. It may be arrested by a mustard leaf on the epigastrium. If the face is red or cyanotic, the pulse full, the coma deep, the temperature low, the respiration labored, venesection is indicated, and from twelve to eighteen ounces of blood should be taken from the arm at once. It will do no good unless done within a few hours of the attack. In hospital cases I have seen this cause great relief, and believe I have seen it save life. It should be employed much more freely than it is in cases of cerebral hemorrhage. That it is a harmless measure is shown by its general use in the last century for all sorts of diseases. The reaction against its abuse has led to its neglect in cases where it is indicated. The cases in which it does harm are cases of weak heart, small pulse, and general anæmia. Placing a tight bandage around the limbs near the body will prevent venous blood from returning, and thus take some blood from the brain, and this is a means to be used when bleeding cannot be done. Mustard paste to the legs or back of the neck accomplishes similar results, but serious blisters are too often caused in states of unconsciousness by these means and by hot bottles to make their use advisable. Applications of ice to the head are usually ordered, but it is questionable if they do good. They are, however, agreeable to some patients when consciousness returns and there is pain in the head; hence it is possible that they relieve congestion. No attempt should be made to arouse the patient from the coma or to feed him, and when he begins to recover, all excitement, the presence of several persons, and attempts to talk, should be prevented; and quiet in a darkened room should be preserved. When it seems certain that a hemorrhage is occurring the use of adrenalin hypodermically or by the rectum is advisable. It is also well to cause free purgation by giving two drops of croton oil in a little butter. This put on the tongue will be absorbed in the mouth, and is not likely to cause choking if swallowed. Convulsions may be controlled by chloroform or by chloral hydrate given by rectum in twenty-grain doses.

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[1] American Journal of the Medical Sciences, 1901, vol. cxxii. p. 854.

When the patient recovers consciousness his anxiety should be quieted by reassurance that the danger is over. No attempt at feeding should be made until he feels hungry, and then cold milk only should be given for the first three or four days. If there is difficulty in swallowing a small tube may be introduced through the nose, a spray of cocaine being first used in the throat, and milk given through this. Water may be given freely if desired, and, in cases of nephritis, should be urged upon the patient. In such cases, if there is not a free excretion of urine, an enema of salt solution will be advisable. Heart stimulants should be avoided, and wine, coffee, or tea are not to be used unless a condition of weakness of the heart becomes very manifest. Aconite in small doses frequently repeated is of service, as it quiets the increased activity of the heart. Hypodermic injections of camphor, 2 grains, dissolved in thirty minims of olive oil is the best heart stimulant if one becomes necessary. If the patient is very restless bromide of sodium in thirty-grain doses every six hours may be given, and if sleepless, trional in fifteen grain doses may be used. Tepid sponge baths with alcohol are very quieting and refreshing. Great care should be taken to keep the body clean. The bladder should be emptied by catheter every three hours to prevent soiling of the bed, and a daily enema should be used to clean out the rectum. Castor oil, or aloes, or cascara may be given to keep the bowels open. Pads of oakum or cotton may be used to prevent pressure, and the position must be changed every little while to relieve restlessness and prevent bed-sores. An air-bed or water-bed is rarely necessary, but may give comfort.

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[1] R. L. Bowles. Stertor and Apoplexy. London, 1891.

It has long been the custom to give five or ten-grain doses of iodide of potassium three times a day to assist the absorption of a clot. Those who believe that it will accomplish any result may continue to use it.

If it is apparent that the stroke of apoplexy is due to an embolism, or a thrombosis, and the patient is pale and the heart is weak, he should be laid flat in bed, and heart stimulants with vasodilators should be used at once. A hypodermic injection of nitroglycerin, one-one-hundredth grain in whiskey or cognac should be given, and the alcoholic stimulant repeated or given with hot water as an enema. Ammonia may be inhaled and Hoffman's anodyne may be used. Heat should be applied to the body, but the measures recommended in hemorrhage—venesection, purgation, ice to the head, and adrenalin—are contraindicated. If convulsions occur chloroform may be used or chloral in large dose by enema. Absolute rest should be enforced, no effort allowed as the patient recovers consciousness, and then, when he is able to swallow, supporting food, mild stimulation with coffee, hot drinks, milk, and beef tea should be given. These measures are of great value in the slight prodromal attacks which precede a stroke in cases of endarteritis. I have seen very marked and favorable effects from the use of caffeine citrate or the sodium salicylate of caffeine, three-grain doses every six hours. If the pulse tension is high nitrite of sodium in two or three-grain doses every four hours may be used, as it is less sudden and more prolonged in effect than nitroglycerin. It may be given with the caffeine. If an inflammatory reaction follows the embolism, and the temperature rises, and restlessness and insomnia follow, the caffeine must be stopped, and bromide of sodium or chloral hydrate used. The latter in five-grain doses every four hours is of much service in these cases, as it does not increase the arterial tension and yet quiets the brain. Trional is also of service to cause sleep. A rapid relief from the coma not infrequently follows this treatment in cases of embolism or thrombosis as the collateral circulation is stimulated. In the cases where the temperature rises and active encephalitis occurs, ice to the head, antipyretics, such as phenacetin, acetanilid, cool sponging, and laxatives are indicated, but medication accomplishes little.

When it is impossible to make an accurate diagnosis between hemorrhage and thrombosis or embolism it is not safe to employ active medication directed to either condition, lest the damage to the brain may be increased. In such cases it is better to secure absolute rest; to aid the respiration by position on one side; to see that the condition of the heart and pulse is favorable, using either mild sedatives like aconite, or mild stimulants like camphor and caffeine, as they are indicated; to apply ice to the head, and if there is much restlessness to use bromides freely. When consciousness returns quiet should be ensured, a mild light fluid diet ordered, plenty of water given, the skin and bladder kept in order, the bowels evacuated by enemata daily, and sedatives, bromide or trional, used if needed.

In cases of mild onset without loss of consciousness the same degree of rest is to be secured as in severe cases, and for at least two or three weeks after the attack, no matter how slight, no physical or mental exertion should be allowed. A light diet, plenty of water, and mild laxatives should be used, and stimulants avoided. The subsequent treatment should depend largely upon the condition which is suspected or determined. As the majority of cases of slight attack are due to thrombosis in diseased arteries rather than to hemorrhage, vascular dilators and caffeine, with or without a small amount of digitalis, or sparteine may be cautiously tried, and if found to be of benefit used more freely. If they cause headache or other premonitory symptoms they must be discontinued. In cases where spasm or convulsive movements have occurred the use of bromide is indicated.

In all cases the treatment of the paralytic condition following an apoplexy is really expectant. It is impossible to hasten the process of repair in the brain, and time alone can bring about a spontaneous recovery.

. During the two or three weeks after the attack the patient must not be allowed to sit up or to make any effort for any purpose. He must be fed, his bladder emptied by catheter, the bedpan used for his evacuations, and he must not turn himself in bed when bathed. Cold applications to the head should be kept up for a week or longer if they are agreeable to the patient. By the end of a week the diet may be increased and easily digested solid food may be given. A variety of fluids, such as lemonade and mineral waters, may be given, and weak tea and coffee may be allowed, but no stimulants, unless the case is one of thrombosis. The position of the patient must be made as comfortable as possible by the use of cushions supporting the arm and leg, and their position may be changed often. Bathing with warm water and alcohol is very soothing, and may be done twice a day. No medicine is needed during this stage unless it is some sedative at night. Constant encouragement is needed, and the hope of recovery must be stimulated. At the end of two or three weeks in light cases, and at the end of four or five weeks in medium cases, the patient begins to draw the leg up in bed or to straighten it out when it has been passively flexed. After such motions have been possible for a week, but not sooner, the patient may be lifted to a chair and allowed to make attempts at standing with the aid of two persons or one person and a crutch. It is not well for a patient to exert himself in this way too soon or too long, for fear of a relapse. And it does not hasten his recovery to allow him to try. The paralyzed arm should be supported in a sling.

The cases are few where it can be accurately determined that there is a surface hemorrhage which can be removed by surgical interference. When this is the case there should be no hesitation in undertaking the operation. Thus in a case in which McBurney trephined for a traumatic hemorrhage causing aphasia and right hemiplegia, the subsequent progressive recovery would undoubtedly have been impossible had the clot not been removed.[1] In subcortical hemorrhage no operation can be of any service, for the clot cannot be reached except by an extensive incision into the brain, which in itself causes serious and permanent symptoms.

The treatment of the hemiplegia, which should be begun at the end of the second week, is by massage of the limbs daily or twice a day for fifteen minutes at a time, by passive movements of the limbs, and by the application of faradism to exercise the muscles, especially the extensors of the wrist, the muscles moving the fingers, and the flexors and extensors of the ankle and knee. These muscles should be exercised for a minute at a time, and the entire application should take half an hour. The facial muscles do not require electrical treatment, as the instinctive movements exercise them. It should be distinctly understood that the effect of electricity is limited to exercise. The use of galvanism to the head has long since been abandoned as useless and possibly harmful. Any device which will enable the patient to make voluntary motions, and thus exercise the muscles by act of will, is far more useful than electricity. Thus as soon as any power appears in any muscle, the patient is to be urged to use it. He can do this if the action of the opposing muscle be performed passively or by a mechanical device. Thus grasping a bulb which contains a spring will exercise the flexors of the fingers while the extensors are still unable to open the hand; pressing on the pedal of a sewing machine will exercise weak flexors of the ankle while the extensors are still unable to lift the toes. Such devices not only serve to exercise the muscles but also to occupy the patient's mind, which is usually devoted wholly to the consideration of his state. Muscle beating helps some patients, and may be combined with massage Warm applications followed by cool affusions sometimes stimulate the circulation in the limb and help the strength; and as soon as walking is possible it should be allowed, and stepping over obstacles, and careful voluntary motions of the leg should be practised, and the attention should be directed to the act of walking so as to prevent a hemiplegic gait from being acquired. The same training as has been used by Fraenkel in the treatment of locomotor ataxia is very useful in cases of hemiplegia. (See page 327.)

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[1] Brain Surgery, Case XVIII.

The hemianæsthesia may occasionally be benefited within a week by the application of a faradic current to the limbs, the faradic brush being preferred. If it has no effect, however, within a month of the onset there is no treatment which will restore sensation, and faradism usually increases the subjective numbness which annoys the patient.

The hemianopsia cannot be treated in any way, and it is often well not to call the patient's attention to it, as little by little it is disregarded. I have seen a number of patients who had bilateral homonymous hemianopsia but were not aware of the fact.

The aphasia is open to treatment by education in some cases, but not in all. Practice in reading, in reading aloud, in naming objects and in writing is sometimes successful in recalling the lost memories and powers of expression. I have had patients who learned to read again by the use of the letters of the alphabet printed on little cards. I have known patients who acquired a new language before they relearned their own tongue. I have known patients who could sing before they could talk. I have known patients to carry about a little dictionary and to find in it the word they could recall by sight but could not utter, and thus to convey their ideas. If no attempts at re-education are successful the deaf-and-dumb language should be acquired, and it may interest the patient to teach him this from the outset, showing him large printed letters and letting him form a new association of ideas between them and the position assumed by the hands. If hemiplegia attends the aphasia the well hand may be used. The cerebellar symptoms usually subside spontaneously, but obstinate vertigo can sometimes be relieved by the use of bromide.

Post-hemiplegic tremor or athetosis is not benefited by any form of treatment.

The mental symptoms cannot be treated. But the patients can be kept from excitement, from emotional strain, from mental effort, and must have cheerful surroundings and mild forms of amusement. It is better for the general welfare of educated and active persons to allow them some definite mental occupation, and if new interests cannot be started it is better to permit some apparent resumption of professional or business life, and visits of former associates, than to allow them to drift into a state of mental depression over their apparently useless condition. Cheerful companionship, entertaining reading by a good reader, games, the dictation of correspondence, and the general interests of life may afford sufficient occupation. And eventually travel may be allowed. It is well for a person who has had one attack to avoid extremes of climate and high altitudes. A winter in a dry, mild climate like that of Bermuda, Nassau, California, the Nile, or the Riviera, and a summer in the mountains or by the sea, not exposed to great heat, will prolong life.

CHAPTER XXVII.

THE CEREBRAL ATROPHIES OF CHILDHOOD.

Infantile Hemiplegia, Diplegia, Feeble-mindedness, Imbecility, Idiocy, Blindness, Deaf-mutism, Epilepsy.

MALDEVELOPMENT OF THE BRAIN.

The brain may be injured or diseased in fœtal life, or at the time of birth, or during infancy, and in consequence may fail to develop in a normal manner. When this occurs a condition of cerebral atrophy is found after death. The atrophy may be limited in its extent to a few convolutions, or may involve one lobe of the brain, or may affect the entire brain in greater or less degree. The symptoms produced by cerebral atrophy are numerous, but patients are easily assigned to one of three groups of cases, in accordance with the chief feature presented, though in some patients the symptoms of all the groups are found.

Clinical Groups. The first group of cases is characterized by spastic paralysis, which may be hemiplegic or diplegic in type. In the diplegic form both legs may be affected, or both arms as well as both legs. The paralysis is often accompanied by aphasia and by athetosis, and not infrequently by sensory defects, by imbecility, and by epilepsy.

The second group of cases is characterized by mental defects of greater or less degree. These may vary from absolute idiocy to feeblemindedness, or the patient may show merely some peculiarity of conduct, indicating a lack of the highest powers of attention, and of reasoning, and of self-control. Epilepsy may develop in these cases.

The third group of cases is characterized by sensory defects: either hemianopsia, or total blindness, or deafness, and consequent mutism. In these cases also imbecility and epilepsy may develop.

Frequency. Cerebral atrophy is not rare. I have records of 400 cases. Of these 274 belonged to the first group, 219 having hemiplegia, and 55 diplegia. Among these patients 100 were aphasic; 89 had athetosis, and 90 had epilepsy. In 120 some degree of mental defect was apparent. To the second group 111 patients could be assigned, as the mental defects were more marked than other symptoms. Sixty of the second group were epileptics. To the third group 15 patients could be assigned, 7 being blind, 3 having hemianopsia, and 5 being totally deaf from birth. Six of these patients were epileptics, but none was imbecile.

Etiology. In some cases prenatal influences are responsible for the maldevelopment of the brain. Osler reports a case of hemorrhage found in the brain of a fœtus, the mother having died of typhoid fever at the sixth month of pregnancy. Cotard has reported a similar condition, the mother having received an injury. Any severe disease in the mother may affect the development of the fœtal brain. Syphilis in either parent may prevent a proper evolution of the child's nervous system. Sachs holds that the frequency with which infants of neurotic, epileptic, alcoholic, and insane parents present cerebral defects is proof that prenatal influences are potent causes of cerebral palsy and idiocy. It has been thought that cerebral thrombosis and encephalitis may occur in the fœtus.

Many cases date from birth. In these some evidence of traumatism can usually be obtained. Thus in 177 of my 400 cases there was a history of difficult labor, either a long protracted labor, or a malposition requiring manual delivery, or instrumental delivery. In several cases labor was premature, and the child was kept alive with difficulty. In two cases the patient was a twin. In many cases the child was born asphyxiated. In all these cases it is probable that meningeal or cerebral hemorrhage occurred during delivery, and that the pressure upon the brain or some injury to it prevented a normal development.

In some cases the symptoms appear suddenly in a normal infant during the early years of life, the first three years being the period of greatest liability. Among my patients eighty-two developed symptoms during the first year, fifty-eight during the second year, twenty-eight during the third year, the remainder later. Some cases are traumatic. Twenty-two of my patients had suffered from severe falls during infancy. Other cases were due to cerebral hemorrhage, embolism, or thrombosis, or to an acute encephalitis. Any of these conditions may occur as a complication of the infectious diseases of childhood, and hence cerebral palsies or idiocy may date from an attack of measles, diphtheria, whooping-cough, cerebro-spinal meningitis, pneumonia, scarlet fever, or typhoid fever. There was a history of one of these diseases in twenty of my patients. Meningitis and hydrocephalus are common causes. In a great many cases the occurrence of a convulsion is the cause assigned; but whether the convulsion is the first symptom of the lesion, or whether in a convulsion some vascular rupture causes a lesion of the brain, is never a matter of certainty. Forty per cent. of my patients had an initial convulsion.

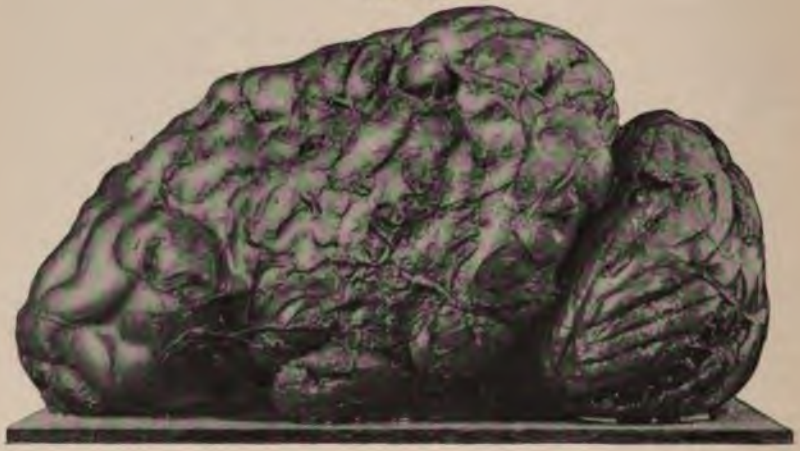
There are also cases in which no cause can be found. An arrest of development of the brain may occur at any time during the first twenty years of life, and then produce symptoms.

Pathology. Records of the pathological condition found in all the three groups of cases described have been compiled, collections of cases with autopsies having been made by numerous writers.

The lesions found are various in kind, in origin, and in situation, but a careful study elicits two conclusions: First, the difference in the clinical types is due to the varying situation of the lesion rather than to its varying nature; secondly, that the various processes of disease have, as a fairly uniform result, a condition of atrophy with sclerosis of the brain, which we may term sclerotic atrophy.

In the first clinical type the sclerotic atrophy involves the motor area of the brain—*i. e.*, the central convolutions bordering the fissure of Rolando and the cortex of their immediate vicinity, and involves also the motor tract arising from this part of the cortex, and usually the basal ganglia as well. (See Fig. 214.) Sometimes the lesion is confined to the basal ganglia. In the second type the sclerotic atrophy involves the anterior portion of the brain, and sometimes the entire hemisphere to a greater or less extent. (See Fig. 215.) In the third type the sclerotic atrophy involves the posterior and lateral parts of the hemispheres.

FIG. 214.



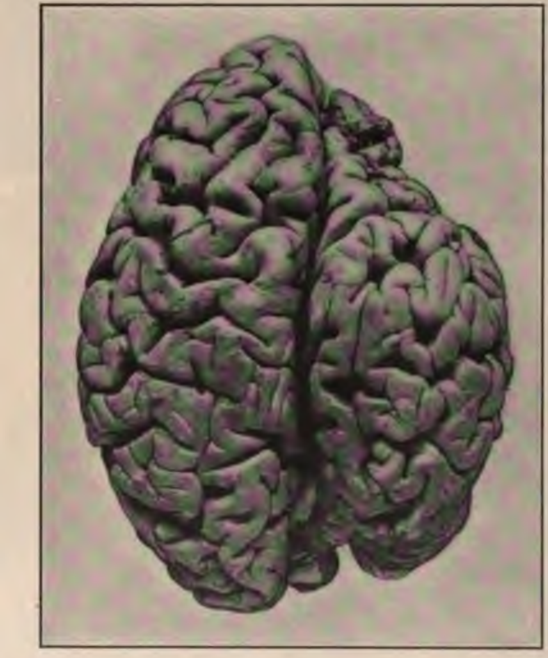
Sclerotic atrophy of the cortex supplied by the middle cerebral artery in a case of diplegia, idiocy, and epilepsy. Both hemispheres were affected. (Peterson.)

It is not surprising that the variation in the situation should produce varying symptoms in view of the facts of the localization of brain-functions already stated. That there should be a limitation of the sclerotic atrophy to certain lobes or regions—to the frontal, or central, or occipital, or parieto-temporal regions—in various cases, has led to the conclusion that the origin of the disease lay in some interference with the blood supply of the part affected, since it has often been evident that the atrophy was limited to the region nourished by blood reaching it through one arterial trunk. And this theory has been rightly supposed to explain the pathogenesis of these cases. Whether thrombosis or hemorrhage has occurred is not always easy to determine.

It has been stated that the various processes of disease have, as a uniform result, a condition of sclerotic atrophy. This fact is borne out by the following *résumé* of pathological findings in 343 cases which have been gathered from the records of Kundrat, Audry, Wallenberg, Osler, Wilmarth, Féré, Henoch, Hirt, Fowler, Schultze, Sachs, Richardière, Bourneville, Fisher, and from recent American and foreign journals. The conditions found were as follows:

Porencephalus, a localized atrophy or agenesis, leaving a cavity in the cerebral hemisphere, which may be deep enough to open into the ventricle, 132 cases. Figs. 216 and 218 show this condition.

FIG. 215.



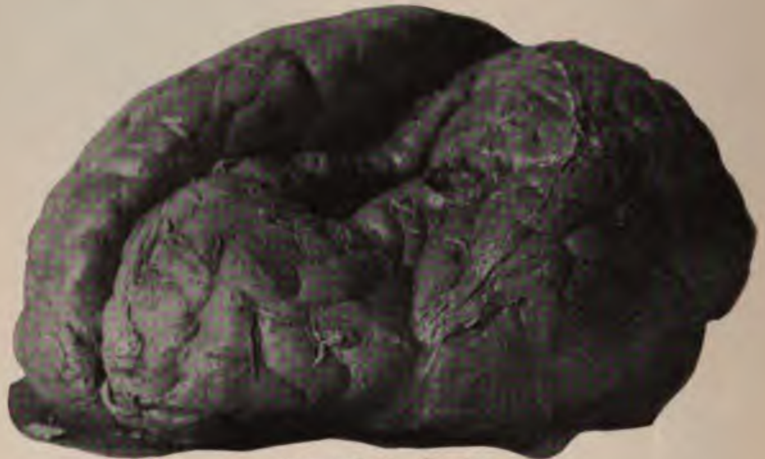
Congenital maldevelopment of the right hemisphere of the brain, with sclerotic atrophy. The child was hemiplegic, idiotic, and epileptic.

Sclerotic atrophy, an atrophic condition of the brain with an increase of connective tissue and disappearance of the nervous elements; affecting both hemispheres, or one only, or a part of one only, or limited to small areas in various parts, 97 cases. This is the terminal result of encephalitis (*q. v.*) and often results from maldevelopment from unknown causes acting on the fœtus.

Maldevelopment and apparent atrophic condition of the minute structures of the hemisphere, chiefly cortical, the cells resembling those of a newborn child, but with no apparent gross defects in the brain, 32 cases.

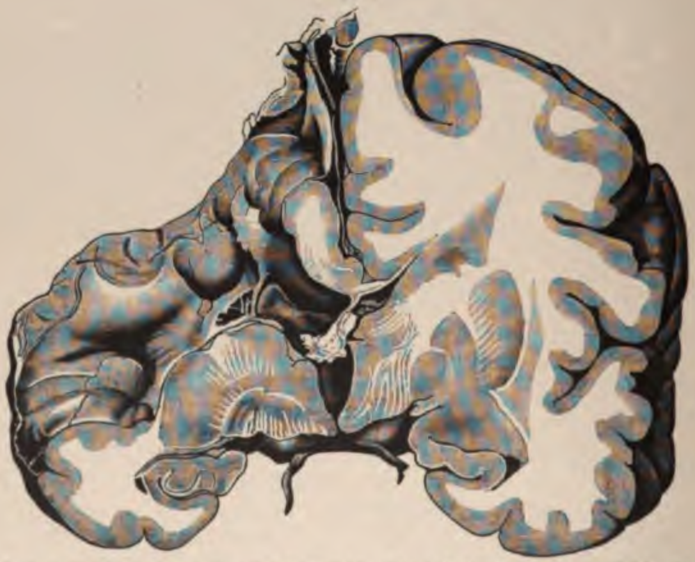
Atrophy, consequent upon the condition of softening produced by embolism or thrombosis, and limited in extent to certain arterial districts of the brain, 23 cases. Fig. 214 shows this condition.

FIG. 216.



Porencephalus. (Lloyd.)

FIG. 217.

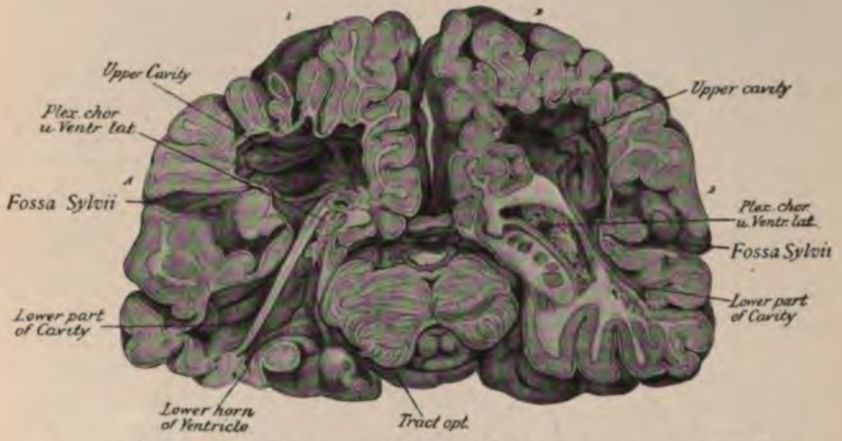


Frontal section through a porencephalic brain. The left hemisphere is normal. The right hemisphere is atrophied *in toto*, and has a cavity in the motor region which extends downward into the ventricle. The basal ganglia are atrophied. This condition is always congenital. (Shattenberg.)

Meningo-encephalitis, a condition shown by thickening and adhesion between the pia and the brain, with destruction of the cerebral cells and atrophy of the cortex, 21 cases.

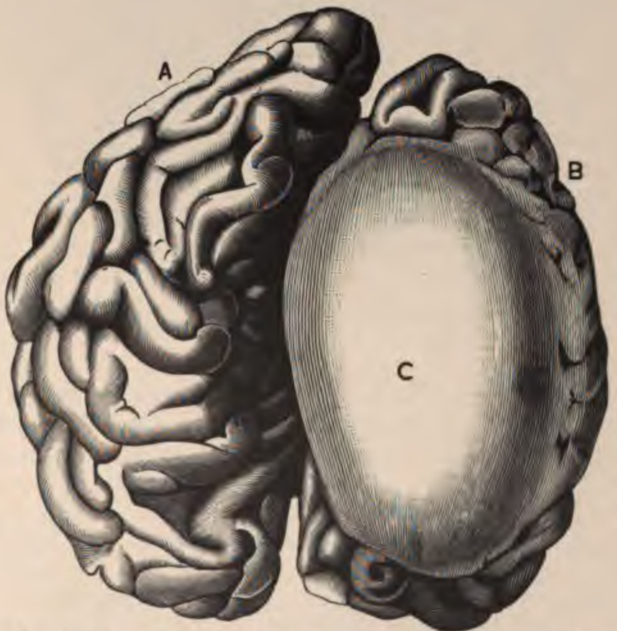
Cysts lying on the brain and producing atrophy by pressure, or associated with atrophy due to the original lesion of which the cyst remains as a trace, 14 cases. Figs. 219 and 220 show this condition.

FIG. 218.



Extensive bilateral porencephalus. The cavity within the brain communicated with the external surface at *A*. (Richter, Arch. f. Psych., xxxii.)

FIG. 219.



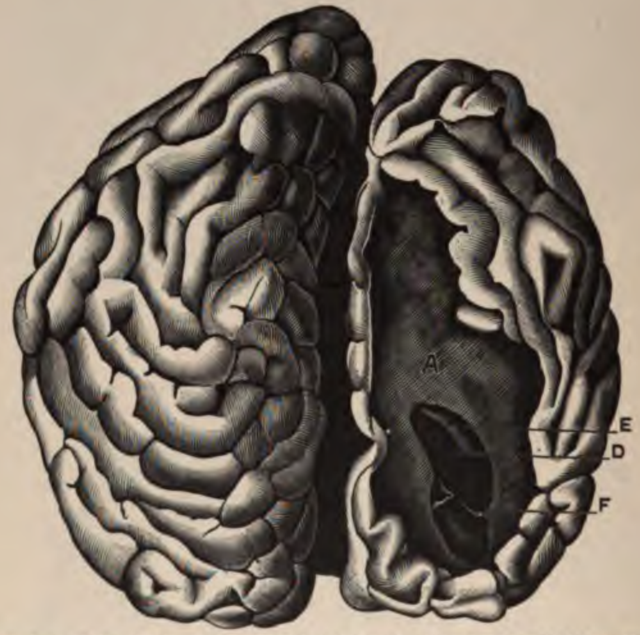
Superior surface of a brain of a congenital imbecile who had hemiplegia and epilepsy. The entire right hemisphere is atrophied. C is the arachnoid, which was thickened and formed the wall of a cystic cavity in the hemisphere. (Ferraro.)

Hemorrhage on or in the brain, as shown by the remains of a clot, or by hæmatin staining of a cyst, of the pia, or of the sclerotic tissue, 18 cases.

Hydrocephalus with extreme dilatation of the ventricles, so that the brain tissue is reduced to a mere wall about the cavity, 5 cases.

Hydrocephalus is an accumulation of serum in large amount in the cranium. If the serum collects in the meninges it compresses the brain and is associated with atrophy. This is termed *external hydrocephalus*. It is a rare condition, secondary to meningitis or to maldevelopment of the brain, and not infrequently the fluid is poured out to fill the space left between an atrophic brain and the cranium. The common form of hydrocephalus is *internal hydrocephalus*, that is a distention of the ventricles of the brain by an exudation of serum into them.

FIG. 220.



Superior surface of the brain of a congenital imbecile. The arachnoid being removed, the porencephalic cavity is displayed. The cortex is wholly defective over the upper frontal and parietal lobes, and the cavity in the hemisphere opens into the lateral ventricle at D, in which the choroid plexus E is seen. (Ferraro.)

Internal hydrocephalus may be acute or chronic. The acute form is always due to meningitis, either simple or tuberculous (see Chapter XXXVIII.). The chronic form may be secondary to a mild meningitis or ependymitis, the original disease resulting in an adhesion within the ventricles, which closes one of the foramina and prevents the free flow of serum from the lateral ventricles outward through the aqueduct of Sylvius and the fourth ventricle and the foramen of

Magendie into the space about the brain. It may also be secondary to any disease in or outside of the brain which compresses the base and obstructs the free exit of serum from the ventricles. Thus tumors of the brain usually cause hydrocephalus. It may also be secondary to syphilitic disease in the brain or meninges, and many congenital cases are due to inherited syphilis, with closure of the aqueduct or foramina. Chronic internal hydrocephalus is in many cases a congenital condition, and is very frequently associated with rickets. It is considered a primary condition in cases where no cause can be ascertained, and it is in these cases that the most extreme distention of the brain is observed. The fluid appears to be secreted by the ependyma of the lateral ventricles, and may vary in amount from a few ounces up to five or six pints. It is identical with the cerebrospinal fluid. It gradually distends the ventricles, dilates the passages between them, and compresses the brain. The brain becomes anæmic and atrophies, so that finally a mere, thin capsule of brain tissue is left about the ventricles. The gradual compression and atrophy of the brain interfere with the function of the tracts passing through it, and hence gives rise to spastic diplegia, to sensory defects, to mental deterioration, and imbecility. The distention of the brain causes a distention of the skull, giving rise to the characteristic hydrocephalic skull, in which the sutures are separated, the frontal and parietal bosses bulge outward, and the head is of enormous size. The softening of the bones may be the earliest sign of a beginning hydrocephalus. Holt states that the average head at birth measures 14 inches in circumference, and at one year measures 18 to 19 inches. A rapid enlargement beyond these limits during infancy is suggestive of hydrocephalus. The enlargement may progress slowly for several years, or may cease at any time, spontaneous arrest of the condition being observed in about 6 per cent. of the patients. Ninety-four per cent. of these patients die before the age of seven years. Attempts to drain the ventricles do not result in an arrest of the secretion, and hence are almost uniformly unsuccessful.[1]

Unilateral hydrocephalus was found in one case in the pathological records.

It is evident that the common condition in all these cases was an atrophy of the brain.

The maldevelopment of the brain is usually accompanied by a state of microcephalus, all the measurements of the skull being reduced as compared with a normal head. In many such children great asymmetry of the skull is found. In other cases the head is unusually large. A large head may contain a microcephalic brain, the space about it being filled with fluid. The asymmetry becomes more noticeable as the child grows up. The following table shows the measurements of the average normal skull and the limits of physiological variation in an adult. In cases of cerebral atrophy these limits are passed.

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[1] See Brain Surgery, Chapter VII.

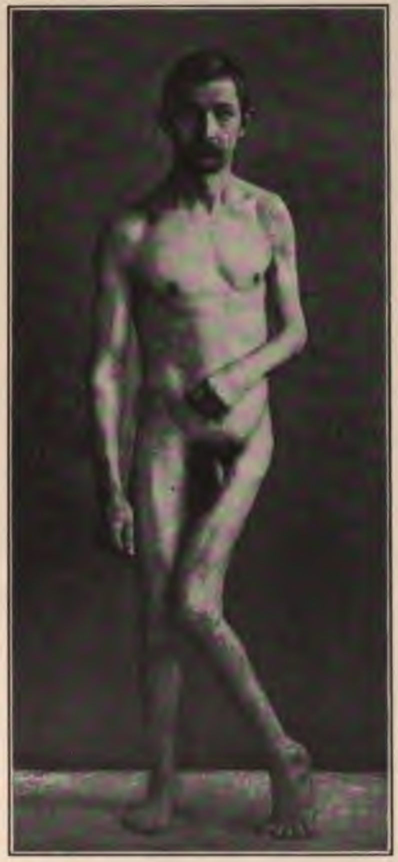
TABLE OF MEASUREMENTS OF THE SKULL. (PETERSON.)

	Average of normal male skull.	Limits of physiological variation.
Circumference	52.0 cm.	48.5 to 57.4 cm.
Volume (rough approximation)	1500.0 "	1201.0 " 1751.0 "
Naso-occipital arc	32.0 "	28.0 " 38.0 "
Nasobregmatic arc	12.5 "	10.9 " 14.9 "
Bregmato-lambdoid arc	12.5 "	9.1 " 14.4 "
Binauricular arc	32.0 "	28.4 " 35.0 "
Antero-posterior diameter	17.7 "	16.5 " 19.0 "
Greatest transverse diameter	14.6 "	13.0 " 16.5 "
Length-breadth index	82.2 "	76.1 " 87.0 "
Binauricular diameter	12.4 "	10.9 " 13.9 "
Facial length	12.37 "	10.5 " 14.4 "

I. **Symptoms of Cerebral Spastic Paralysis.** The first group of cases present symptoms of paralysis. This may be unilateral or bilateral.

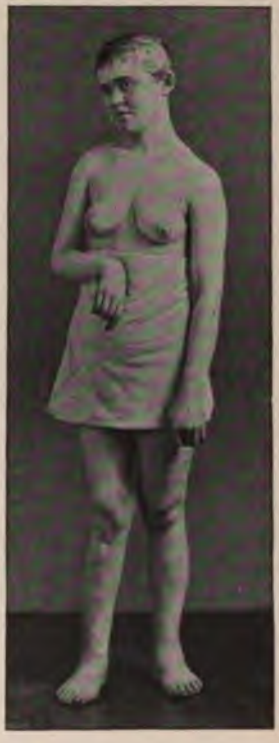
Hemiplegia. In the cases which develop a unilateral paralysis the characteristic features of hemiplegia as seen in adults are evident. This may date from birth or it may develop later. If it develops in infancy it usually begins with a series of convulsions, which are often general, but may be unilateral, and are attended by high fever, 102° to 105° F., and nausea, vomiting, and headache, with delirium. The onset is followed by a period of unconsciousness of varying duration, from an hour to two or three days, the average being twelve hours. The child may die in such an attack. But there is usually a gradual improvement in the paralysis after the active manifestations of the onset have subsided; and, finally, a stationary condition remains, in which the face is but slightly affected in its voluntary or automatic movements; the speech is acquired very slowly, or is slowly regained, if it had been lost; the arm is quite seriously paralyzed, the fingers being stiff and awkward, and sometimes being in constant slow involuntary motion which is called athetosis; the leg is weak, and the knee and ankle are held rigid, so that the child limps in walking, and sometimes has a club-foot. A slow improvement is the rule. As time goes on the child learns to help himself in many ways, and it is possible for the gradual growth of the limbs to be attended by increasing power. In all cases the paralyzed limbs are found to be affected in their growth and development, so that they are smaller, colder, stiffer, and weaker than the others; they are often blue and cyanotic. The reflexes are exaggerated on the paralyzed side. The electrical reactions are not qualitatively changed. The sensation is normal. This condition remains through life as a permanent defect, and although the division of contractured muscles or tendons, and the application of ingenious apparatus, may correct deformities and make the paralyzed parts fairly useful, and although the application of electricity to the muscles chiefly affected may increase their nutrition, and thus prevent contractures which come from the unbalanced strain between the various muscles, yet a perfect recovery does not occur. The degree of spontaneous recovery possible in any case cannot be determined for several years. But in many cases while some trace of the hemiplegia remains there is so much improvement that the patient is capable of earning his living. I have several such patients who, though unable to use the hand freely, and always obliged to limp in walking, are able to occupy places as clerks or messengers, or have been able to go through college, and even to study a profession. It is only in the cases in which weak-mindedness and epilepsy occur that the condition is a hopeless one. But even when the final state is not one of helplessness these patients are extremely liable to other nervous

FIG. 221.



Hemiplegia, with contractures. The patient had suffered since the age of two years. (Curschmann.)

FIG. 222.



Spastic hemiplegia from angiolithic sarcoma of the brain, the symptoms dating from five years of age. (Dercum.)

diseases. Thus several such persons under my observation have suffered from chronic neurasthenia and hysteria. One of them developed attacks of mania of short duration but frequent recurrence, not unlike the psychical epileptic equivalent, and, though an educated lawyer, has required asylum care for several years past.

Spastic Rigidity; Athetosis. The especial characteristics of this form of hemiplegia are the spastic rigidity of the affected limbs with marked contracture, and the athetosis. The contractures lead to various deformities about the ankle and wrist, especially to club-foot. Fig. 221 shows these conditions. Athetosis may occur when the hand or foot are quiet. When the child attempts to move the hand or leg unexpectedly involuntary slow motions occur which prevent the intended result, or the limb is thrown into a sudden state of rigid immobility. Athetoid movements on the paralyzed side are set up by any voluntary act on the well side. These athetoid motions begin soon after the onset as a rule. In one case of birth palsy they did not appear until the fourth year and increased until the seventh year, remaining when I saw the child at the age of twelve years. If the athetoid movements affect the face and tongue, grimaces are constant and speech is interfered with. Athetosis has been present in 30 per cent. of my cases. (See Fig. 194.)

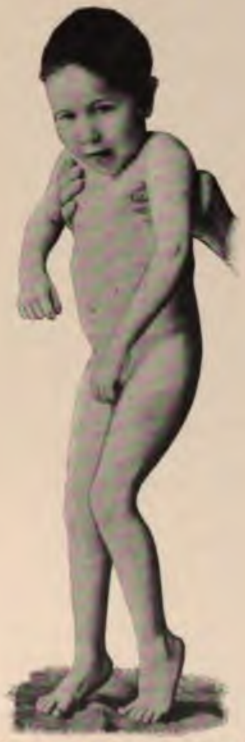
In some cases the athetosis is the most marked symptom. Thus in the case of a boy, aged eleven years, who had been born asphyxiated after a face presentation, and was revived with difficulty, a peculiar type of athetosis had been present since the age of ten months. The motion was limited to the left side and was present in the face, in the hand, and in the foot. It was not constant in this case, nor was it started by an effort. But it came in attacks, twelve or more in a day, each lasting several minutes. The motion was a quick, involuntary athetoid movement, which he could not control. It was not attended by a loss of consciousness, or by a sense of alarm, or by any numbness. He had no paralysis whatever, no nystagmus, no disturbance of vision, no headaches, and was bright and active mentally. The attacks were first noted during sleep, but as he grew older became more frequent in the daytime, and no treatment had any effect upon them.

In another case, also dating from birth, the athetosis affected the entire body, so that any effort was attended by contortions of the face and by peculiar crowing sounds which interfered with talking and eating; by constant violent movements of the arms and legs, so that no voluntary act could be performed, and walking was impossible. The boy, at the age of ten years, was perfectly intelligent and not paralyzed, but was in a constant state of active motion without any volitional power. It was impossible for him to sit upright in a chair or to stand. He could only lie in bed supported by pillows, and was even fed with difficulty. The athetosis ceased during sleep. I have recently seen a second similar case equally distressing in a young man, aged eighteen years, whose entire life had been spent in bed.

Choreic movements more sudden, rapid and violent than athetosis may appear in the paralyzed limbs and remain as a permanent symptom. Such a condition was noticed in twenty-one of my two hundred and seventy-four cases.

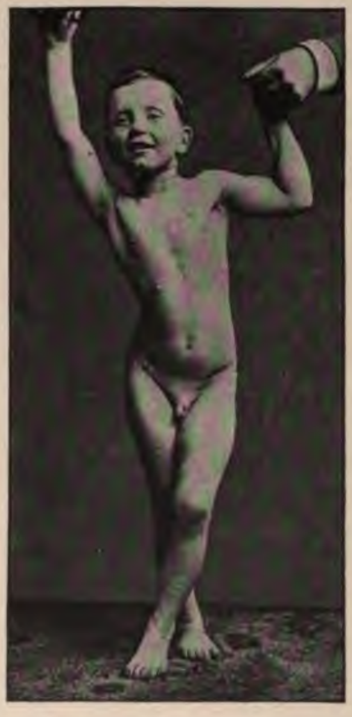
In one of my cases there was a total **paralysis of the tongue** with right hemiplegia, and the child at the age of twelve years was still unable to articulate. Food was pushed back into the throat by the finger, and then swallowed. This child had learned to read, but was weak mentally and epileptic.

FIG. 223.



Marked spastic paraplegia; walking or standing alone impossible. (Dercum.)

FIG. 224.



Spastic paraplegia; crossed-legged progression. (Dercum.)

Aphasia is a common accompaniment of right hemiplegia. In my 274 patients, 130 had right hemiplegia, and in 100 of these aphasia was present. It is usually of the motor type. The children understand what is said to them, can acquire a knowledge of the names of objects, and may learn to read, but cannot be taught to talk, or if they acquire language, do so very slowly and imperfectly. I have known children of the age of twelve years to talk like a child of three years. There is, however, as a rule, gradual recovery of speech, and it seems likely that in these cases the right hemisphere takes the place of the left in developing this function. This is often aided by training the child to write with the left hand.

Diplegia—Little's Disease. In many cases dating from birth a condition of bilateral paralysis of arms and legs, or of the legs alone, is found as soon as the child is born. This is termed diplegia. It becomes more apparent after the first few weeks when attempts at voluntary motion are made. It is attended by a rigidity of the muscles which is noticed in the legs, and there is an increase of the reflexes. The infant may have difficulty in sucking and may have to be fed with a spoon.

Diplegia may however develop slowly, and not become apparent for some months. In some cases, especially after the infectious diseases, it may not appear till the age of three years. In these cases as the baby grows its body is found to be stiff, especially in its back and legs, the latter tending to be adducted and extended. This is noticed in adjusting the diapers. Later when attempts at walking are made the adduction and inward rotation of the thighs may cause an overlapping of the knees and feet, there may develop a pointing of the toes which gradually becomes a fixed equino-varus, so that the heels can not be put on the ground, and the inability of the child to balance becomes more noticeable. Any attempt at standing increases the stiffness of the back and legs. Sometimes the back is so rigid that the child cannot sit up on a chair, but has to be kept in a lying position. As the child grows the condition becomes more noticeable, and sometimes the child is bedridden and never learns to walk. In other cases the child acquires a spastic gait and helps itself by its hands, as they are often not affected, and so is able to get about. Others improve slowly but steadily, and finally walk without a cane, but stiffly.

In the severe cases the arms are rigidly adducted and flexed, and the hands are contractured and useless. (See Fig. 223.) In such a condition athetoid movements are universal. The tendon reflexes are always exaggerated, the muscles of the limbs are poorly developed, but there are no bed-sores, and there is no loss of control of the sphincters. There are no sensory symptoms. These children frequently have mental defects, and epilepsy is likely to develop.

Strabismus is a not uncommon symptom, and difficulty of articulation, or of swallowing may be present. Oppenheim has called attention to the timidity of these children and to the fact that sudden noises cause them to jump or even throw them into convulsions.

In a few of my cases the symptoms have been exclusively cerebellar in type. The child has been unable to sit up, to balance itself on its feet, or to learn to walk. In one such case dating from birth, the child was still absolutely helpless at the age of nine years, although intelligent and not paralyzed. In another the same condition was present at the age of five years, but was attended by feeble-mindedness. This child died of measles, and Fig. 203 shows the unilateral atrophy of the cerebellum found.

In many of the cases difficulty in learning to talk has been noticed, the child acquires words slowly, and sometimes even at the age of six years is unable to frame a sentence or to speak clearly.

Epilepsy. In the majority of cases both of hemiplegia and of diplegia epileptic attacks begin soon after the paralysis; though sometimes the interval is several months or even several years. Two-thirds of the cases eventually develop epilepsy.

The fits in organic epilepsy are more frequent and severe than in idiopathic epilepsy. I have one patient who has had as many as twenty convulsions daily for weeks at a time. These fits do not destroy life, but of course render life a burden. They are different from the fits of idiopathic epilepsy in being of a character which indicates local irritation of the cerebral cortex. (See page 422.) They usually begin in one hand with closure of the fist, or pronation of the hand, attended by a sensation of tingling; the contraction of the muscles is clonic and the spasm extends up to the elbow, and finally the shoulder-joint is thrown into motion, the entire arm moving in the convulsion. The spasm then extends either to the neck and face, which is drawn to the paralyzed side, or it spreads down the trunk and involves the leg, which shakes or is violently extended with clonic spasms of the toes. Such a spasm may last for a few minutes, and then gradually cease, leaving the muscles paralyzed and the side numb for several hours. Or it may extend to the other side of the body, in which case, as a rule, consciousness is lost for some minutes, the patient falls, bites the tongue, froths at the mouth, and subsequent to the attack sleeps heavily for an hour or more, waking dazed, and often unable to talk or to think for some hours. In many cases the attack begins in the leg and extends to arm and face. In other cases the reverse is the order of extension of the spasm; the point of greatest irritation in the cortex being indicated by the part of the body first affected. In some cases head and eyes turn toward the paralyzed side at the beginning of the fit.

In other cases the epileptic attack begins with sensory rather than with motor symptoms, the initial numbness and tingling always accompanies and may precede the local spasm. But sometimes it is a visual aura, a light, or color, or figure seen, with which the attack begins. In other cases it is a sound, a noise, or a bell, or a voice which is heard. In other cases it is an odor or a taste which is perceived. In all these conditions a local spasm or a general convulsion follows the aura, indicating that the irritation of the brain which has started in a sensory area, the chief seat of disease, has radiated outward and has invaded the motor centres. (See page 435.) Mental auræ are also observed, consisting of a dreamy state, a sort of second consciousness, when the mind seems to watch its own processes without being able to modify them. The mental aura may be a sudden fear, or fright, or distress, pleasant ideas being rarely present, though not unknown.

Attacks of an active maniacal kind or of double consciousness may occur without a convulsion; these are the so-called "psychical epileptic equivalents" of an attack.

All these forms of attack are of very great interest, as they indicate quite faithfully the area of brain in which the disease is located, and form the guide to the surgeon in cases which are open to operation.

II. **Symptoms of Mental Defects.** The second group of cases presents mental defects rather than physical symptoms. There may be an entire lack of mental development from the outset. This is termed *congenital idiocy*, and is due to congenital defects of the brain or to some arrest of growth soon after birth. There may be an *acquired idiocy*, from some disease occurring during infancy or childhood, such as injury of the brain from trauma, or after convulsions, or due to some of the infectious diseases.

A maldevelopment of the thyroid gland leading to cretinism and mental weakness is not attended by brain lesion, and should not be classed with idiocy.

Idiots are wholly lacking in the power of acquiring ideas or concepts, and cannot be trained to keep themselves clean.

If the degree of mental defect is less marked the mental state is described as one of imbecility. An imbecile can acquire simple ideas, can recognize persons and objects, can say a few words, but is incapable of acquiring any of the higher conceptions which are the basis of knowledge and of conduct. Imbeciles are usually helpless if left alone, and are often ugly in disposition, destructive and filthy. The difference between an idiot and an imbecile is one of degree only, and no sharp line can be drawn between them.

There are other patients with mental defects, of a higher type of mind than imbeciles, but nevertheless abnormal. These are classed as weak-minded. The child may be slow in learning to walk; may seem unable to fix its attention upon anything continuously; may be exceedingly active, in constant motion—the activity being, however, aimless; may throw things about, or tear things up, or put everything into its mouth; may be very difficult to manage because of its inability to retain and combine impressions with sufficient power to reason upon them, and may therefore be incapable of appreciating the meaning of punishment, if this be inflicted. Such children may have good powers of perception, may recognize persons and objects, show pleasure at bright colors, or music, or caresses, but fail to show evidence of thought in the sense of reasoning power, judgment, or self-control. Some patients constantly drool at the mouth, cannot be taught cleanly habits, and are manifestly imbecile. Other patients are quite bright in many directions, may even be precocious, show talents in music, or drawing, or fondness for mathematics, designing, and languages, yet are apparently unable to appreciate moral ideas, cannot be taught to tell the truth, are cruel and bad, will not control any of their impulses, and so are the distress and despair of parents and teachers. It is those mental qualities which are the product of the highest evolution that have failed to develop in this class of cases. The final result is that they have to be taken care of all their lives, either at home or by attendants, being incapable of supporting themselves or directing their conduct. Many of them have epilepsy.

III. **Sensory Defects.** The third group of cases show sensory defects, usually blindness or deafness, although a loss of taste and smell is occasionally observed. These symptoms are likely to escape observation until the child reaches the age of two or three years. Patients belonging to this class may present no motor or mental defects, though they may be the subject of epilepsy. On several occasions I have been consulted by parents who have brought children to me supposedly the subjects of epilepsy in whom I have detected one or more of these sensory defects. Thus, in three cases a condition of homonymous hemianopsia was discovered which was undoubtedly congenital, each child having grown up without any appreciation of the defect of vision. In all of these cases there was a corresponding flattening of the skull over the occipital lobe, and all the patients were suffering from epilepsy. Moeli[1] has described three such cases in which porencephalus was found in the occipital lobe, and Henschen[2] and Freund[3] have also observed such cases. In several patients whom I have seen total blindness has been found. It is not always easy to determine whether this is due to a primary atrophy, or lack of development in the optic nerve, or whether it is due to atrophy of the occipital convolutions. Both conditions have been found post-mortem in many cases. If the condition is due to atrophy of the optic nerve an ophthalmoscopic examination will reveal this condition.

There are other cases in which the patients are deaf. If a child has never heard, or lost its hearing before it has learned to talk, it becomes mute. The majority of cases of deaf-mutism are undoubtedly due to lesions in the ears or to lesions of the auditory nerves upon the base of the brain. There are a few cases, however, on record in which an atrophy of the temporal convolutions has been attended by deafness.

Loss of smell and taste have been produced by atrophic conditions of the uncinate convolutions upon the base of the brain. These are usually traceable to basilar meningitis. They very often escape observation, though in any case where the child is known to put all sorts of objects into the mouth and be willing to eat offensive things, a suspicion of a loss of taste is awakened.

A localized atrophy in the convolutions of the brain may also occur from disuse. Thus, in a number of cases recorded where patients have lost the sense of hearing and of sight in early life, as, for example, from basilar meningitis, cerebro-spinal meningitis, or lesions of the ear, or eye, the function of the corresponding portion of the brain has not been awakened, and as a result of absolute disuse an atrophy of these convolutions has been discovered when the patients have died late in life. This was the condition found in the brain of Laura Bridgman, the temporal and occipital convolutions being rudimentary in size, while the sensorimotor convolutions about the fissure of Rolando were enormously developed. This girl lost sight and hearing at the age of two years from a basilar meningitis, but developed remarkable powers of observation through the sense of touch; and of expression through motor action. In two other cases reported by foreign observers the same condition has been found. Hence the localized atrophy due to failure of development in limited areas of the brain after disuse of the corresponding organs is proven in man as well as established by the experiments of von Gudden.

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[1] Archiv. für Psych., xxii., 2.

[2] Henschen. Path. des Gehirns, cases 32 and 35.

[3] Freund. Wien. med. Woch., 1888, No. 32.

Epilepsy is a common condition in these cases of sensory defect. The attack not infrequently begins by an aura of a sensory nature. It may be limited to an attack of *petit mal*, or it may go on to an attack of *grand mal*. If the atrophy of the brain is unilateral the epileptic attack may be of a Jacksonian nature.

Prognosis. The prognosis in cerebral atrophy is most unfavorable. The underlying pathological condition is permanent, even though it is not progressive, and there is no hope of improvement or of recovery. The paralysis, the mental defects, and the sensory defects cannot possibly be relieved. The epilepsy may be held in check to some extent by the use of bromides, the number of fits being reduced, their severity somewhat mitigated. But they cannot be stopped.

Treatment. The treatment of an acute attack of apoplexy in childhood does not differ in any way from that occurring in the adult. The same care must be taken in diagnosis, and the same measures adopted as in the conditions of hemorrhage or thrombosis already considered.

The treatment of the condition of infantile paralysis and of idiocy consists in rendering the life of these patients as happy and comfortable by proper surroundings and agreeable attendants as possible. This can best be done by securing for them a country home away from excitement and away from the family. Parents too often expend more care, attention, and money upon these chronic invalids than upon their healthy children who suffer in consequence. It should be made clear to them that in so doing they accomplish little and are unjust to those who will better repay their care. Education by those who are skilled in the training of the weak-minded accomplishes something, and is to be recommended if parents can afford the expense. The treatment of the epileptic condition is the same as that of ordinary epilepsy by the use of bromides; but in these cases the effect of the drug soon wears off, and I prefer not to use any medical treatment.

Surgical Treatment. When a patient belonging to one of these clinical types is presented to the neurologist and the question is asked, Can surgical treatment benefit him? it is evident that a serious problem is opened. In these cases the disease is at a standstill and does not threaten life, and yet is hopeless from the medical standpoint. Surgical treatment is not free from danger, but if it offers any relief almost any risk is justifiable. The brain is still capable of great development in infancy and youth. Can such development be aided by an operation? In many cases the epileptic attacks are of such frequent occurrence that any risk might well be taken if they could be surely stopped.

Any solution of the problem of operative treatment must be based upon two considerations: first, the pathology of the cases; secondly, the results of experience when such operations have been done.

First, from the records of the pathology already given in 343 cases it is evident that a state of cerebral atrophy was present in the majority which nothing could remove, and it is apparent that many of the conditions were of such a nature as to be wholly unaffected by any operative interference. A porencephalic cavity filled with cerebrospinal fluid is not likely to be benefited by any enlargement of the intracranial space or by the abstraction of the fluid. In several cases operated upon the withdrawal of this fluid has been followed by sudden collapse and death.[1] On the other hand, there are conditions, such as maldevelopment of the cortex without gross lesion, in which it is possible that anything which may stimulate latent powers of growth, or may remove those influences which interfere with development, might result in improvement. It is to be remembered that the brain is capable of growth and development until the age of fourteen years, if not longer, and, granting that a stimulus to its growth may be given during childhood, the arrest of development in many cases might perhaps have been prevented. And there are cases of cysts lying upon the cortex whose removal may relieve pressure and allow a proper growth.

The study of the pathological conditions, therefore, does not absolutely contraindicate operative interference, although it makes it clear that the lesion in the majority of the cases is one that cannot be improved by any means. If we admit that porencephalus, atrophy from vascular lesion, meningo-encephalitis, and hydrocephalus are incurable, and that hemorrhages cannot be diagnosed early enough to warrant the removal of the clot before it has caused atrophy from pressure, we have 193 cases out of 343 in which operation would have been futile. This leaves 150 cases of sclerotic atrophy, maldevelopment of the cortex, and cysts, in which it is barely possible that an operation, if it relieved pressure or stimulated brain growth might have had some effect. It is unfortunate that we cannot make an exact pathological diagnosis from the clinical symptoms. Therefore, one cannot affirm that in any particular case a removable cyst is present or a condition of pressure which may be relieved. The operation must in all cases, therefore, be exploratory, and this should always be understood by all concerned.

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[1] Cases of Bullard: Boston Medical and Surgical Journal, February 16, 1888. Hammond, New York Medical Journal, August 12, 1890. One of my own.

There is one further fact which requires a word of explanation. In many of these cases inspection shows a very decided irregularity of the skull. It has been thought by some observers, especially Lannelongue, in France, that an early closure of the sutures and fontanelles in infancy may prevent a proper expansion of the skull, and thus produce a pressure on the brain, preventing its growth. These authors regard the atrophy as secondary to compression. This view I consider erroneous. The skull does not unite as long as there is any pressure within it. It is the cerebral defect due to disease which prevents the brain from developing and arrests its normal growth and internal pressure. The bones close because the natural expansion has ceased. The skull does not cause pressure on the brain. This is evident in cases operated upon, for no signs of compression of the brain have been found in these cases. The theory, therefore, upon which operations of craniectomy have been done is not borne out by the facts.

Secondly. The results of experience of surgeons in the treatment of these cases by operation has not been very favorable.

The operation of craniectomy has been done by many surgeons during the past few years, and the results have been reported by several of them, notably by Lannelongue, Keen, Bullard, Oppenheim, Frank, Hammond, Horsley, Agnew, and McBurney. Many other operators have reported single cases, and my own experience in connection with McBurney is extensive.[1]

Lannelongue reported, in 1891, that he had operated on 25 patients, chiefly microcephalic or epileptic children, with but one fatal result, and that there was an improvement in many cases. Lannelongue's operation is to make a long, curved incision through the scalp and skull from the temporal ridge backward for six to ten inches, and crack the side of the skull outward, leaving an interspace of an inch or more in width which fills up with fibrous connective tissue. Thus he relieves the supposed compression of the brain. It is unfortunate that these cases were reported soon after the operation, so that, although the surgical result was not fatal, we know nothing of the effects of the operation on their brain development. In a recent article by one of Lannelongue's assistants the intimation is given that little was accomplished.

In my own collection of cases, 50 in number, including 15 in my own experience, the result cannot be said to be very encouraging.

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[1] For an analysis of these operations see the Medical Record, January 23, 1892. Brain Surgery, Chapter III.

Of 50 cases 16 died during or soon after the operation. Death was due in 11 cases to shock from sudden evacuation of fluid from a large cavity in the brain. Thus, in one case operated upon for me by McBurney, in which the child, a girl of eleven years, presented only a slight degree of right hemiplegia, with severe attacks of hemiepilepsy and no imbecility or aphasia, the entire left frontal lobe was deficient, its place being occupied by a cyst. The exposure of this on opening the dura was attended by laceration of its wall, as it was adherent to the dura, and the flow of serous contents was followed by immediate collapse and death. In 10 cases reported by others a similar result occurred. In some cases death has been due to excessive hemorrhage from large brittle veins in the pia or in the walls of a porencephalic cavity. Here ligature was impossible, and compression equally so. In some cases death has been due to exhaustion following the operation, which has necessarily been long. It is evident, therefore, that these operations are much more dangerous than the ordinary operation for trephining, because of the existence of pathological conditions which cannot be foreseen or provided against.

In the thirty-four patients who have survived the operation a certain amount of improvement has been reported in twenty-five, and in nine no apparent result was noticed. The improvement has consisted in a relief of the paralysis, but not in its disappearance; a marked relief from the athetoid movements, but no cessation; an improvement in the gait; an improvement in the mental capacity, and a change from imbecility to a condition of weak-mindedness, but no complete recovery or normal development of the mind; a diminution in the frequency and a mitigation in the severity of the epileptic attacks, but no absolute cure.

In several of my own cases nothing abnormal has been found in the brain at the operation, and it has been supposed that the lesion was of a microscopic character. In two of these cases the subsequent history has shown considerable improvement, which must have been wholly independent of the operation, and awakens the suspicion that such improvement of a spontaneous kind may have been erroneously ascribed to the operation by partial observers.

In three of my cases a thin organized blood clot containing fine connective-tissue filaments adherent to the dura in one case and to the brain and cortex in two cases was discovered. It was possible to sponge and scrape away this clot in all these cases, though some hemorrhage followed from the rupture of the new capillaries in the organized mass. These were all cases of several years' duration, hence the clot was not recent in any. In all these cases there was an apparent marked improvement. In a number of my cases cysts have been found—sometimes lying on the cortex, in the pia, and causing a depression of the cortex; in other cases within the brain, being in part covered by the cortex; in some cases deep in the white matter, being discovered only by the exploratory needle. It has usually been possible to remove these cysts, the walls being carefully dissected away without rupture. It was found that if they were merely evacuated the fluid soon returned, and a second operation was necessary. This occurred in two cases. When the cyst lay deep in the white matter and could only be discovered by puncture it has not been always possible to remove the wall, and in these cases they probably refilled, as no improvement followed. The best result has been obtained in the cases where a cyst could be removed as a whole. In one case a cystic mass exactly like honeycomb occupied the entire lower two-thirds of the anterior central convolution, and could not be removed entirely.

In some cases thick, firm, connective-tissue scars, or porous bands have been found in the cortex, in one case as firm as cartilage, in another case calcified in part. These were probably the result of hemorrhages or of an area of softening. Their removal was attended by severe hemorrhage, in one case making it necessary to postpone twice a completion of the operation. In neither of these cases were the mental symptoms and epilepsy for which the operation was undertaken at all improved.

This experience leads me to be very cautious about urging an operation in this class of cases. The dangers are many. The prospect of relief is very small. Cure cannot be promised. Improvement in a small proportion of the cases only is the best result which can be expected.

CHAPTER XXVIII.

ENCEPHALITIS.

Acute Hemorrhagic Cortical Encephalitis. Polio-encephalitis Superior and Inferior. Cerebellar Encephalitis.

Etiology. That an acute inflammation of the brain may occur as an independent affection has been established by Strümpell, Leichtenstein, and Oppenheim. It is probably an acute infectious disease, as it has all the characteristic signs of one. It has been known to occur in the course of scarlet fever, measles, influenza, pneumonia, erysipelas, whooping-cough, mumps, diphtheria, ulcerative endocarditis, otitis media, and septicæmia. It is not necessarily accompanied by purulent meningitis, though it may be. In most of these conditions the characteristic organism of the original disease has been found in the foci of inflammation in the brain. Ptomaine poisoning has been ascribed as a cause, also alcoholism and poisoning by carbon dioxide. It is still a matter of dispute whether an injury of the head not attended by fracture or meningitis can set up an acute inflammation of the brain. Bruises in soft tissues are rarely followed by inflammation, but may predispose an organ to the invasion of bacteria. I have seen abscess to occur under these conditions, and believe it to have resulted from such an inflammation. The disease occurs chiefly in children and young people, but may develop in adults.

Varieties. The inflammatory process is usually limited in its extent in the brain, and is not diffuse. It is sometimes only to be found by microscopic investigation. It is sometimes wholly within the domain of one arterial branch. In other cases it has been known to affect symmetrical parts of both hemispheres, and in a few cases disseminated foci of inflammation have been found. The inflammation may be found in both gray and white matter, and is not limited to either. It may affect any part of the brain. A number of different forms have been described, depending upon the location.

Thus, Strümpell's first cases were chiefly cortical in the motor area, and were called acute infantile cerebral palsy, or acute hemorrhagic cortical encephalitis. Later this condition was found to occur in adults.

In the cases collected by Wernicke and named by him polio-encephalitis superior the lesion was located in the gray matter lining the aqueduct of Sylvius. These cases are not different from acute ophthalmoplegia.

There is a form limited to the nuclei of the motor cranial nerves of the tongue and face, termed polio-encephalitis inferior, or acute bulbar paralysis, and causing all the symptoms of chronic bulbar paralysis (Chapter XXXIII.), but with acute onset and rapid course.

Lastly, a form limited to the cerebellum has also been described as a separate disease.

Pathology. The lesion in all these cases is the same, and the difference in the symptoms is dependent entirely upon the different functions of the parts of the brain affected. Such a multiplication of diseases is an unfortunate weakness of authors who lack the generalizing faculty. The disease has its exact homologue in acute anterior poliomyelitis, and the changes present are the same. The two diseases have been known to occur together. The changes found in these cases in the acute stage are (1) an acute hyperæmia with distention of the bloodvessels, rupture of their walls, capillary hemorrhages in the tissues, and an emigration of leucocytes and small cells. (2) Various stages and degrees of degeneration of the neurones, both cell body, axones, and dendrites being affected, with subsequent secondary changes of wide extent. The exact microscopic changes are identical with those already described in the chapter on anterior poliomyelitis, but are located in the cortex or bulbar nuclei.

After an acute stage the hyperæmia subsides, the clots or diffusely infiltrated red blood cells may be absorbed, and if the degeneration of neurones has not led to the destruction of a large number controlling any special function a gradual recovery ensues and no trace is left. In this respect the disease resembles anterior poliomyelitis, where recovery in many of the muscles at first paralyzed is the rule. But if a number of neurones in any system are destroyed there are permanent symptoms remaining whose character depends on the function of the neurones affected. Thus, if the frontal lobes are affected, imbecility results. If the motor cortex is destroyed a state of hemiplegia remains. If the ocular motor nuclei are affected strabismus and paralysis of ocular movement results. If the facial and lingual nuclei are paralyzed some defect in speech, swallowing, and facial expression remains. If the cerebellum is affected a permanent defect of co-ordination and of gait ensues. If the cord is affected with the brain, some type of poliomyelitis attends the encephalitis.

The permanent result of the pathological process is the formation of small or large areas of sclerotic tissue or of small cysts. The glia cells are multiplied, connective-tissue elements are increased, and a small patch of sclerosis or a state of sclerotic atrophy of an entire lobe of the hemisphere, or any degree between these extremes, may be left as a permanent condition. Thus, disseminated encephalitis of infectious origin is the starting point of many cases of so-called multiple sclerosis. It is not impossible that, as time goes on or as other infections occur these sclerotic patches may extend, but, as a rule, they are latent. These pathological changes in the brain when on the surface are often attended by the lesions of meningitis in the pia and dura. Hence, in the terminal stage an adhesion of the membranes to the cortex is frequently found. In a few fatal cases of recent origin thrombosis of the venous sinuses has been observed. The occurrence of leucocytosis during life will distinguish this class of cases from those due to vascular lesions only.

Symptoms. The symptoms of encephalitis may be divided into two classes: first the general symptoms of the infectious disease, and secondly the local symptoms of the particular region of the brain affected.

The general symptoms develop acutely. After a day or two of indefinite feelings of malaise, attended by vertigo and headache, and in children by extreme fretfulness, there is a sudden chill, attended sometimes by vomiting and by a convulsion, followed by a state of stupor or coma. The temperature rises rapidly to 102° or 104° F., the pulse is rapid but regular, the respiration normal, rarely of the Cheyne-Stokes type. In the state of stupor there is restlessness and delirium. This stupor may deepen into a coma which may never be recovered from. If the patient does not die in the coma he gradually recovers consciousness, but delirium and even acute maniacal excitement are not uncommon for a week or more, and have been known to continue with fever for twenty days. The recovery of consciousness is slow, and the mind is not clear for some days. The fever subsides by remissions in the morning, and its attendant symptoms of general constitutional disturbance gradually pass away. Leucocytosis is observed throughout. A day or two after the onset some local symptoms become evident, and then the diagnosis of hemiplegia, or of ophthalmoplegia, or of bulbar palsy, may be made.

The local symptoms in the first type of case are aphasia, or hemiplegia, or monoplegia, and these are not infrequently combined at the outset. The aphasia, as a rule, passes away, but some degree of hemiplegia or monoplegia remains. Thus, in one of my patients, while total aphasia and right hemiplegia occurred at the onset, a paralysis with athetosis of the right arm was the only permanent symptom. In another case which developed suddenly during convalescence from cerebro-spinal meningitis, a left hemiplegia with mental weakness remains.

In another type of case the local symptoms may be sensory rather than motor, some form of word-blindness or word-deafness being a permanent result, or hemianopsia, as in a case of Furbringer. It is probable that this is the origin of deaf-mutism in some cases.

In other cases a state of imbecility develops, and the child naturally born, and bright before the attack, grows up deficient in intelligence and in the higher powers of the mind.

In some rare cases, as in one of my own, the local symptoms point to the cerebellum as the site of the inflammation. A child of three years, able to walk and feed itself, was left after such an acute attack, which came on after measles, with a great degree of ataxia in the hands and an inability to balance itself on its feet, to stand or to walk or to sit up without the aid of its hands. In one such case of Bethe cerebellar lesions were found.

In all these types of cases epilepsy of the cortical variety is a common sequel, and may be the only permanent symptom remaining. It is my belief that many cases of so-called idiopathic epilepsy, especially those that are characterized by a uniform sensory or motor aura, originate in acute encephalitis during infancy or childhood.

In another type of cases (polio-encephalitis superior of Wernicke) the local symptoms are confined to the eye muscles; there is ptosis of one or both eyes; the eyeballs cannot be moved voluntarily together; there is some form of strabismus; there is nystagmus, and in a few cases optic neuritis has been observed. There is always intense vertigo with this condition, and hence a staggering gait is sometimes noticed. I have seen but one such case. The patient was a physician, and recovered entirely within a year. Weakness, tremor, and ataxia of the limbs, facial paralysis, difficulty in speech and in swallowing have occurred in the severer cases. In adults, especially in patients who are alcoholics, this type is more common than the hemiplegic type. It may develop with few constitutional symptoms and even without fever. In this respect it resembles acute anterior poliomyelitis in adults.

In still another group of cases the local symptoms are those of an acute bulbar palsy. Speech, swallowing, and respiration are affected, the various characteristic signs of glosso-labio-laryngeal paralysis appear simultaneously, and in these cases a fatal result is more common than recovery. These have been termed polio-encephalitis inferior. But the lesion is not always limited to the gray matter of the pons and medulla, and when it extends to the tracts passing through them the symptoms may be widespread. Thus various forms of alternating hemiplegia and hemianæsthesia or general paralysis may be the result.

The combination of cerebral with spinal symptoms has already been mentioned.

The course of the case in all these various types is usually one of gradual improvement, and a final recovery with a few local symptoms remain. Such are monoplegia, sensory defects in the visual field, paralysis of one or two ocular muscles, or epilepsy. In some cases all the local symptoms disappear, and then it is evident that few neurones were actually destroyed by the inflammation. In other cases a most irregular combination of symptoms such as nystagmus, defective articulation, tremor, and some form of paralysis, remains, and the terminal state is identical with that of multiple sclerosis.

Prognosis. The prognosis during the acute onset must be guarded, as death sometimes occurs. It will depend upon the severity of the symptoms, especially on the occurrence of convulsions and upon the depth of the coma, the degree of temperature, the condition of the pulse and respiration, and the power of the individual to combat an acute infection. The prognosis after the acute stage has passed is favorable for recovery, and even serious local symptoms may pass away. The prognosis is better than in cerebral hemorrhage, embolism or thrombosis, as the focus of disease is usually smaller and there is not such a gross lesion. Recurrence of the disease has been seen in three cases only (Dinken, Wiener, and Oppenheim).

Treatment. The treatment divides itself into the care of the patient during the acute stage and the treatment of local symptoms later. At the outset purgatives may be given and ice applied to the head, and antipyretics may be used freely, antipyrine, quinine, salicin. Leeches to the mastoid region are to be applied excepting in very anæmic persons, and may be repeated daily during the febrile stage. Hot baths with mustard may be given to children, and hot foot baths to adults. When the stage of onset is over the various local symptoms are to be treated in the same manner as if the case were one of apoplexy, or of ophthalmoplegia, or of bulbar palsy, or of infantile spinal paralysis.

CHAPTER XXIX.

MENINGO-ENCEPHALITIS. PARESIS. DEMENTIA PARALYTICA.

PARESIS or dementia paralytica is a diffuse degenerative disease of the cerebral cortex characterized by mental, motor, sensory, and vasomotor symptoms, of progressive course and fatal ending.

Etiology. Males are much more liable to paresis than females. It is a disease of early adult life, the majority of cases developing between the ages of thirty and forty-five years; but no age is exempt, as cases have been reported both in childhood and in old age. Mott[1] has recently reported 22 cases of juvenile general paresis, and has collected 75 other cases. It is evident, therefore, that the disease may develop in children. In the majority of these cases hereditary syphilis was found to be the cause. It is a post-syphilitic disease in about 60 per cent. of the cases, though statistics gathered from 54 different authorities by Mott[2] gave a varying percentage from 11 per cent. to 94 per cent. But, as in locomotor ataxia, it is not due to an active syphilitic process, since the pathological lesions are not peculiarly syphilitic, and it is not arrested by antisyphilitic treatment. The interval between the syphilitic infection and the onset of paresis is usually a considerable one—from five to fifteen years. Mental strain and anxiety are the chief exciting causes of the disease. The tremendous efforts to attain financial and social success, the enormous responsibilities undertaken by ambitious men, the effort to carry through large projects, and the ceaseless work day and night, with no rest and little sleep, which are characteristic of the present time in our large cities, are the chief factors in the rapid increase in the number of cases of paresis. It is admitted by all that it is a disease of the more highly civilized, the more mentally active type of men. Hence, the higher classes are more liable than the lower, and those who lead an agricultural life are almost exempt. Peterson states that in Egypt the disease is very rare, though syphilis is prevalent there. The Mohammedans do not use alcohol. Alcoholism and excess in sexual indulgence are certainly additional factors in its production. A certain number of cases seem to be traceable to injuries of the head and to sunstroke.

Pathology. The disease begins with changes in the vessels, hyperæmia of the pia and small cortical vessels, increase of nuclei in the vessel walls, occasional stasis in the capillaries, and exudation of serum into the lymph spaces. As it advances, a formation of fusiform dilatations of the vessels and the development of fibrils of connective tissue between the vessel walls and the surrounding neuroglia occur. In the neuroglia at the same time a marked increase of the nucleated cells, with numerous branching processes is in progress, numerous spider cells developing throughout the cortex, around the vessels and lymph spaces, and about the nerve cells. This increase of neuroglia goes on rapidly, producing a diffuse sclerosis of the cortex, which is followed by retraction of the tissue leading to atrophic shrinking. The entire brain takes part to a lesser degree in this process. In the cerebral tissue at the same time there is in progress a degeneration of the finest nerve fibrillæ and of the branching processes of the nerve cells. This begins in the tangential fibres of the first layer of the cortex, but later involves all the layers of cells. A swelling, chromatolysis, with hyaline or fatty degeneration, vacuolization, pigmentation, and final atrophy of the cell bodies takes place. The formation of cystic cavities throughout both gray and white matter is often observed.

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[1] Archives of Neurology of London County Asylums, 1899, vol. i. p. 250.
[2] Loc. cit., p. 169.

Mott,[1] whose investigations are most reliable, believes that the starting point of paresis is a degeneration in the neurones of the cortex. This is attended by the production of choline (see page 28) which can be found in the cerebro-spinal fluid in paretics. Choline is a toxic substance causing a depression of the heart and a lowering of arterial pressure. Mott believes that its presence causes a venous stasis in the veins of the brain, especially those of the convexity opening into the longitudinal sinus, in which the blood flows contrary to gravity. (See Fig. 207, page 486.) He points out the fact that it is in the domain of these veins that the thickening of the pia arachnoid occurs. The choline accumulates in the perivascular lymph spaces about the veins, causing stasis and inflammation, which are intensified by the action of gravity in the veins that empty into the longitudinal sinus. Œdema of the brain follows, and this is always present in paretic brains.

The final result of these pathological processes in the vessels, neuroglia, and cerebral substance is a gradual atrophy of the brain, so that it weighs much less than normal, sometimes only two-thirds of the normal, the left hemisphere being more atrophied than the right one; it appears shrunken, the convolutions being narrow and the sulci open; the brain tissue is hard, pale, friable, pigmented and on the summit of the convolutions adherent to the pia, which is opaque and thickened. When the pia is stripped off, portions of the cortex come away with it. The thickness of the cortex is seen to be much reduced. A thickened condition of the ependyma of the ventricles is uniformly found. There is usually an increase of fluid within the ventricles and beneath the pia. As a complication the lesions of pachymeningitis with hæmatoma are not infrequently present. All of the changes in the cortex are more marked in the frontal lobes and motor area and about the fissures of Sylvius, but in the later stages of the disease no part of the cortex is normal. In about 10 per cent. of the cases spinal lesions are found in addition to the cerebral lesions. These consist of sclerosis in the posterior and lateral columns of the cord. There are some cases in which the posterior sclerosis is the first lesion to appear, and in these the lesions of locomotor ataxia are well marked, as already described, page 285. In other cases they appear after the condition of paresis has developed, and then they are not so complete. The sclerosis in the lateral columns is usually due to secondary degeneration in the motor tracts after changes in the motor cells of the cortex.

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[1] Archives of Neurology of London County Asylums, vol. i. p. 396.

Symptoms. Paresis begins, as a rule, very gradually, so that many cases are overlooked and others are considered as neurasthenia or dyspepsia for some months, there being present some of the symptoms of one or both of these diseases. After a time the friends of the patient begin to notice that he is not acting naturally, that he is unusually irritable or excitable, is offended or delighted at slight provocation, is variable in his moods, and appears at times inattentive, forgetful, and careless of the proprieties. Soon it is found that his affairs are in confusion, his accounts not properly kept, and that it is impossible to keep his attention concentrated upon important business or upon any train of thought, so that his conversation becomes noticeably fragmentary. He cannot do his work, and yet resents any interference with it, maintaining that he is perfectly well. He will admit some confusion of thought, some cerebral sensations of discomfort, irritability and sleeplessness. His memory for recent occurrences then begins to fail. He neglects engagements; he disregards obligations, sleeps in company, loses his temper, and uses bad language at home or in public, neglects his family, and, though previously moral in conduct, indulges in alcoholic and sexual excesses. By this time it is noticed that his speech is a little thick, indistinct, and hesitating, and a fine tremor of the tongue and lips and possibly of the hands may be detected. The affection is one of fine co-ordinated movements—a cortical ataxia rather than gross paralysis. The pupils are often unequal, or contracted, or dilated, and react very slowly to the light in the early stage; and soon fail to react to light at all, though they react in accommodation.

During this period of invasion headache and sleeplessness are sometimes prominent symptoms, and a gradual loss of weight goes on. From the outset restlessness is noticeable, and after a time it seems impossible for the patient to keep quiet, he wants to take all kinds of exercise, to walk for miles, to go about even when exhausted by fatigue. His intellectual powers are notably weakened, he is no longer attentive, logical, self-controlled, cautious, or careful in conduct, and the change of character becomes apparent both to his family and to his acquaintances. This period of invasion varies from one to three years, and may be interrupted by periods of subsidence of many of the symptoms. It usually goes on to the full development of the disease.

When paretic dementia is fully developed numerous symptoms present themselves—both mental and physical.

The power of sensory perception is not impaired, but the patient is often so preoccupied or indifferent that he does not notice fully or remember the subjects to which attention is directed. His mind passes rapidly from one thing to another, and he does not perceive anything accurately. Thus a patient will forget that he has dined, or neglect to put on some of his clothes, will expose his person, or will commit openly small thefts, will make serious mistakes in his accounts or appointments, merely from a lack of careful perception and consequent impairment of memory. Illusions and hallucinations occur only toward the close of the disease, and are followed by a marked loss of power of sensory perception when the dementia becomes extreme.

The power of logical thought is impaired very early. The patient shows impaired judgment from the outset, takes little notice of important matters, dwells upon trivial things, acts without consideration, and indulges in extravagant schemes, reckless expenditures, and excesses of many kinds which his better judgment would condemn. He may become exceedingly immoral, all restraints of society, religion, and morality being wholly neglected. He becomes profane, obscene, and tipsy. He very frequently develops a delusion of grandeur, says that he never was so well in his life, believes that he is the strongest, the brightest, the most powerful, the most wealthy of men. And he acts at times in accordance with his delusion, undertaking the most extravagant and impossible schemes without any regard for his actual circumstances. Many a patient involves himself and others in financial difficulties by undertaking colossal combinations and extensive business organizations far beyond his powers or means, before it is realized that these schemes are the product of an unbalanced mind. Yet the delusions are not carried out fully in his actions, and he mixes ordinary affairs with them in an incongruous manner. The delusions are not as fixed or as systematized as in paranoia. The development of such delusions is a sure sign, in Meynert's opinion, that an atrophy of the brain has begun.

His emotional state is unstable. He has little control over its manifestations, can be moved to tears or to laughter in the course of an ordinary conversation, can be excited or depressed by suggestions. He is sometimes depressed for days, and feels discomfort, but does not blame himself, and in the midst of the depression may become excited. He is usually sanguine and hopeful, and his emotion is usually in accordance with his delusion, one of exaltation. At times or on provocation the excitement may become maniacal, and outbursts of rage or of frenzy are not infrequent in the course of the disease. Hence a paretic dement is never a harmless member of society, but requires to be watched. He rarely shows any tendency to suicide.

Voluntary action and conduct are affected from the outset; indeed it is by change in conduct that the changes in thought and character are chiefly betrayed, and the weak logical power made manifest. Inconsistency is evident in speech and conduct. Acts are done without consideration; thus impulsive acts, such as stealing, forgery, enormous expenditures, even murder may be performed under sudden excitement or under the influence of a delusion, or in a fit of frenzy. All the ordinary restraints to conduct seem to be removed, and instinct rather than morality is the guide, self-control being greatly weakened.

Self-consciousness appears to be very imperfect. The patient does not appreciate the inconsistency between his previous personality and his present acts; he does not notice the growing anxiety of his family; he regards with indifference loss of means, or even his confinement in an asylum; he is usually so engaged in his imaginary undertakings under the influence of his delusions that he is contented wherever he is. His personality may be changed, and he may imagine himself a prince, a millionaire, a deity. Gradually the consciousness becomes obscured, he recollects little regarding his illness, and as the deeper dementia ensues he becomes indifferent, dull, stupid, and finally almost stuporous. In the final condition of dementia all mental action is suspended.

The physical symptoms are as marked as the mental symptoms.

Motor disturbances appear early. There is restlessness, a tendency to be in constant motion. The patient takes long walks or rides, indulges in unusual exercise, wishes to be going out to theatres every night, to visit friends all the time, and is not content to lead an ordinary quiet life, as before his illness. He has a sense of physical power, and is eager to show his strength, though this may really be impaired. He is very talkative, discussing subjects of which he knows little, or talking at random. Tremor of the muscles appears early, first in the tongue, then in the face and hands, and finally any motion is attended with some trembling. This tremor is followed by inco-ordination, which shows itself in thickness and indistinctness of speech, in irregularity of handwriting, in clumsiness in handling things, and an awkward, unsteady, stumbling gait. The speech is quite characteristic, letters being slurred and words mispronounced, so that any long word or combination of difficult syllables, like "third artillery brigade," cannot be said. In writing the patient will omit letters from words, later may write wholly unintelligible phrases, or even merely make marks on the paper, and yet not appreciate these defects of writing. From the first his handwriting is changed, lines being irregular, letters too small or too large, and badly written. The facial expression becomes blank and inane—no appearance of thought or interest being manifest, but when he talks an excessive play of the facial muscles is noticeable. Paresis shows itself after a time in the limbs, and progresses until in the final stage there is total paralysis, with increased reflex action and loss of all control over bladder and rectum. Finally speech may become impossible. The knee-jerks are either much increased or are lost from the outset.

In the course of the disease epileptiform attacks, attacks of sudden loss of consciousness without convulsions, and attacks of monoplegia or hemiplegia, temporary or permanent, often occur.

Vasomotor disturbances are also present. The face flushes or pales frequently, there are sudden attacks of vertigo and of headache, and of feeling of fulness in the head; the pulse is usually slow, large, and of low tension. In the late stages venous congestions in various organs are found, and hæmatoma of the ears.

Sensory symptoms are less marked than motor symptoms, but as the disease advances anæsthesia or analgesia of the limbs may be found, especially in connection with hemiplegia.

In the last stage trophic disturbances occur, for in the stupid, dirty, helpless state of the patient cleanliness is difficult and the liability to bed-sores and to cystitis is great.

Some cases are complicated by the development of spinal sclerosis, either in the form of posterior or of lateral sclerosis, with its attendant symptoms, and in a few cases the paresis follows the spinal disease.

It is evident that the mental and physical symptoms are very numerous. Many cases show only some of them: others present all the symptoms during the course. In some cases the mental symptoms appear early and are more prominent throughout than the physical symptoms; in other cases the reverse is observed. A few cases begin with epileptiform attacks. In other cases the symptoms begin with a hæmatoma of the dura, causing an attack of apoplexy with aphasia, or hemiplegia, or hemianopsia. These symptoms subside rapidly, so that in a week or two the patient may appear to have recovered. But later the mental and physical symptoms of paresis appear, and then it is evident that the apoplexy was the first sign of paresis.

The course of the disease is slowly progressive, though remissions of one or two years are not uncommon. Its average duration is about three years, though rapid cases may terminate within a year, and some are known to have lasted five or six years. The patients die of exhaustion or of some complicating disease, such as cystitis, or pneumonia, or obstruction of the bowels.

Diagnosis. The diagnosis of paresis is to be made from the combination of physical signs and mental and physical symptoms. The loss of pupil reflex to light, unequal pupils, tremor of the face and tongue, indistinct speech, tremor of the hands, exaggeration or loss of the knee-jerks, are objective physical signs of importance. The mental irritability, excitement with expansive ideas, defects of memory and of self-control are the most important early mental symptoms. Attacks of epileptic or apoplectic nature are the most important physical symptoms.

Neurasthenia may be present in the early stage of paresis, and any or all of its symptoms may then be apparent. But a neurasthenic patient always notices every symptom minutely, describes it fully, and discusses its significance, while a paretic is usually not aware of the fact that he is ill, and does not care to talk about his health as much as about his projects and success. Defects of memory and of speech are rare in neurasthenia, and the neurasthenic presents none of the physical signs of paresis. In a doubtful case it is on the appearance of these signs only that the diagnosis of paresis can be made.

There are some cases of cerebral syphilis which resemble paresis, and it is often difficult in the early stage to differentiate these diseases. Cerebral syphilis usually causes pain in the head, worse at night. If it produces disturbance of speech it is rather in the form of true aphasia, not the tremulous indistinct speech of paresis. There is never any tremor of face and hands, and hence writing is clear and not impaired unless there is agraphia with aphasia, and this differs from the omission of letters and words in writing present in paresis. Optic neuritis is often present in cerebral syphilis, but very rarely in paresis. The emotional state is usually depressed and the patient anxious in syphilis, while it is one of excitement and elation in paresis. Delusions are much less common in syphilis, and are never as extreme as in paresis. The signs of dementia are more marked in an early stage than they are in paresis; the lack of memory, lack of power of reasoning, and lack of self-control appearing early in syphilis and late in paresis. In syphilis there are often physical signs of single or multiple localizable lesions, while paresis is a diffuse disease. The course of the case in syphilis is variable, with much improvement under treatment, while in paresis it is usually progressive.

Multiple sclerosis can hardly be mistaken for paresis; as the nystagmus, tremor of an increasing kind on effort, tremor of the head and trunk, and absence of delusions even when some dementia is present are characteristic of multiple sclerosis.

The dementia of old age and the dementia of chronic alcoholism are not attended by delusions of exaltation and grandeur.

Prognosis. The prognosis in paresis is absolutely bad. Krafft-Ebing declares that he has never seen a recovery in 2500 cases. It must, however, be remembered that remissions in the symptoms occur, especially in the early stage. I have known many patients who have had all the symptoms, to recover for a time under quiet rest in the country, and be able to return to business. Such remission may last for a year or two, during which time the only evidence of the disease may be the inactive pupil, and the slight tremor of the hands, and the exaggerated or lost knee-jerk. I have never known these physical signs to subside, and I have never known a remission to last more than two years. Hence, the prognosis as to recovery is bad.

Treatment. There is no form of treatment which will arrest aresis. In the majority of patients who present a history of syphilis is well to administer a thorough course of inunctions of mercury, followed by large doses of iodide of potassium, this treatment being assisted by the use of daily hot (105° F.) baths for half an hour. In some cases I have seen material benefit as a result, and a remission of some months in the symptoms usually follows such a course. It is absolutely necessary for the patient to stop all business, to rest and to avoid emotional excitement. Hence, it is well to remove him from his home surroundings, and, as he is not fit to travel, it is better to send him to a sanitarium, or to some health resort, or some one of the hot springs, where water treatment can be given. Some forms of hydrotherapy are of service in these cases, but cold baths and douches are to be avoided. A tub bath, or a hot-air box, followed by a shower not below 80° F. and massage daily appears to exert a favorable effect upon the circulation and to quiet many nervous symptoms. When the patient becomes uncontrollable by his family or nurses it is better to commit him to an asylum where he may improve, so that in a period of remission he may be released and allowed to travel. The quiet routine life of an institution, without excitement or responsibility, seems favorable to the subsidence of the symptoms of irritation.

In the later stages of the disease, when dementia is well marked, care in an asylum, or in a home well secluded and provided with skilled nurses, may prolong life for many months.

Some of the symptoms require active treatment. Sleeplessness must be treated by hot baths at night, or massage, or by the use of paraldehyd in 60-grain dose, or trional 20 grains, or sulphonal 15 grains. It is well to give hypnotics with hot milk, as they are more rapidly absorbed and more efficacious. The motor restlessness leads to over-fatigue and heart failure; hence very many short walks in a day, each followed by a short rest, are better than the long tramps which these patients are proud of taking. When the mental excitement becomes very intense, and the patients cannot be controlled by argument, active purgatives, such as compound cathartic pills, aloes, or croton oil, which cause a temporary anæmia of the brain, will quiet them better than sedatives. An occasional use of bromide, or bromide and chloral, or even a hypodermic injection of morphine $\frac{1}{10}$, or hydrobromate of hyoscine $\frac{1}{100}$ grain, may, however, be necessary to allay excitement. A single dose of fluid extract of ergot, one drachm, may have the same effect, but the continued use of ergot is to be avoided.

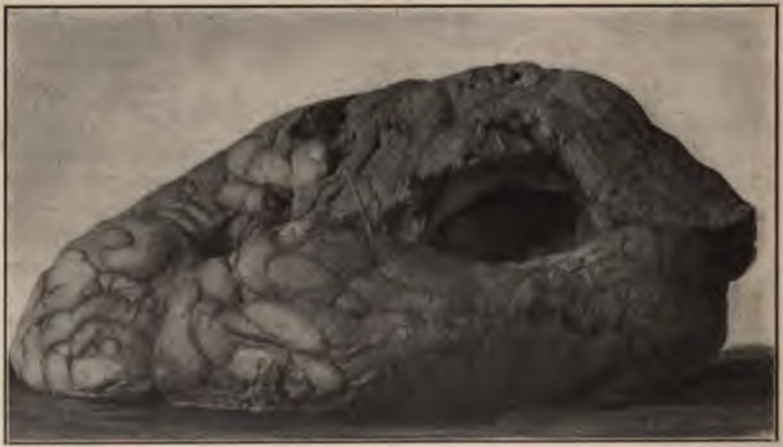
ADDENDUM. Widal affirms that in paresis the cerebro-spinal fluid contains many leucocytes, 125 to 1 c.c., a condition not found excepting in meningitis. He thinks that lumbar puncture and examination of the fluid will establish the diagnosis. Blumer confirms this view.

CHAPTER XXX.

ABSCESS OF THE BRAIN.

Etiology. Abscess of the brain occurs as a sequel to injuries of the head, with and occasionally without fracture. In cases of compound fracture or of wounds in which a septic infection occurs at the time of the injury such abscess formation is to be expected. This is particularly true if a small bit of bone or a foreign body remains in the brain. In cases where the scalp is only bruised it is difficult to understand the source of infection. Von Bergman declares that a simple contusion of the head is not capable of causing an abscess, but I have seen it in several cases where the scalp was not broken, though the bone was fractured. In some cases the abscess follows soon after the injury; in other cases the symptoms do not develop for many weeks or months; and cases are on record where the only explanation for the presence of a latent abscess found unexpectedly at the autopsy was an injury received years before.

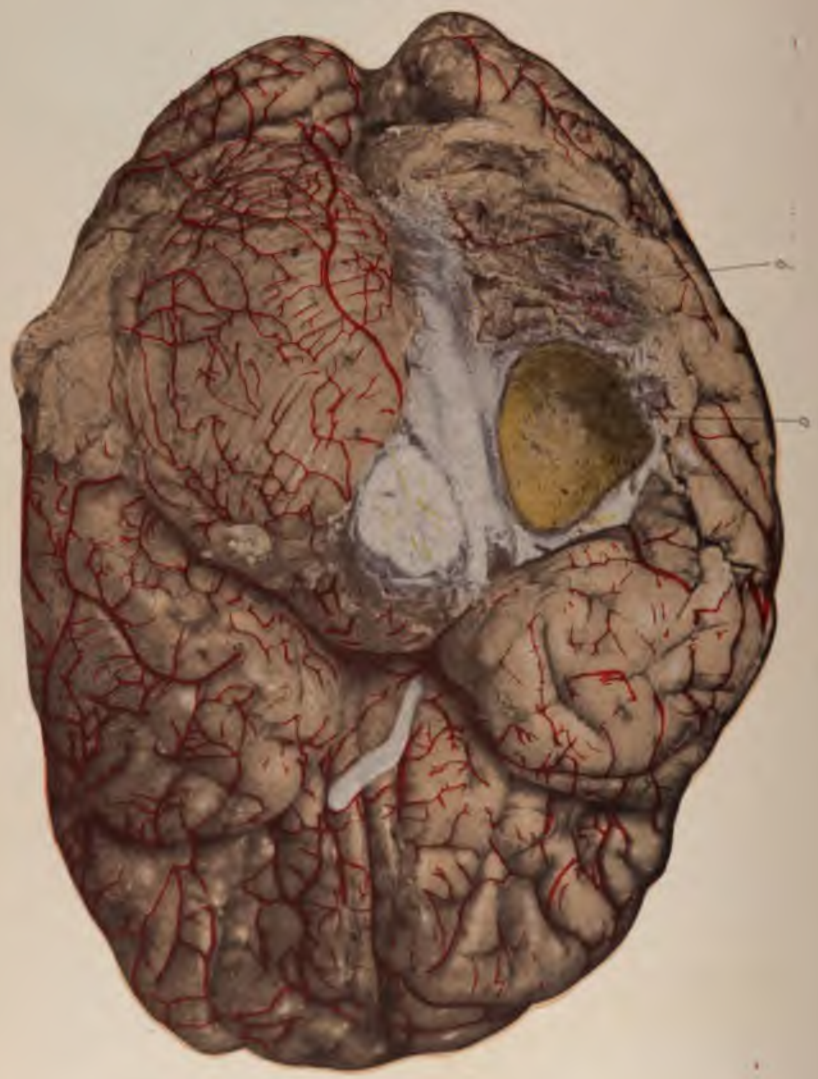
FIG. 225.



Large abscess in the inferior parietal region secondary to fracture of the skull. The thick capsule of the abscess can be seen.

In the case of an infant seen with Poore at St. Mary's Hospital, a fall upon the left parietal bone, causing a laceration of the scalp and an indentation of the soft bone, was followed within two weeks by the development of right hemiplegia with hemianopsia. At the

PLATE XXII.



Abscess of Cerebellum Secondary to Chronic Suppurative Otitis Media. (Bacon.)

The right cerebellar hemisphere has been divided and the lower half removed, exposing the abscess cavity and its wall (*a*) and the area of hemorrhagic softening posterior to the abscess (*b*).

autopsy a large cerebral abscess was found beneath the site of the injury within the white matter of the brain, there being no apparent affection of the cortex for one-fourth of an inch above the abscess. The abscess had a thick capsule and was well defined. (See Fig. 225.) An attempt was made to open it, but the operation had to be suspended on account of the collapse of the patient.

In a second case, seen with McBurney at Roosevelt Hospital, a compound fracture of the superior portion of the left parietal bone, which had healed, had been followed three months later by the gradual development of paralysis of the right leg. At the operation a large abscess was found involving the superior parietal lobule and adjacent portion of the posterior central convolution. In the midst of the abscess a considerable piece of bone was found, which evidently had been driven in at the time of the fracture. The operation was successful. The patient recovered and gradually regained power in the leg.

In a third case, also seen with McBurney at Roosevelt Hospital, a similar compound depressed fracture, with spicula of bone in the brain, had led to the development of an abscess within two weeks of the time of fracture. The abscess cavity invaded the middle third of the motor area, and caused hemiplegia with paralysis chiefly marked in the arm, and here, too, at the time of operation, pieces of bone had to be removed as well as the purulent accumulation around them. The operation was successful and the boy recovered.

In a fourth case, seen with McCosh at the Presbyterian Hospital, a fracture of the skull over the left occipital region had produced well-marked right homonymous hemianopsia. Trephining resulted in the successful evacuation of the abscess, but the hemianopsia remained permanently.

In 22 cases of abscess of the brain observed at the Presbyterian Hospital, 12 have been due to trauma.

The second and probably the most frequent cause of brain abscess is chronic otitis media. A very thin layer of bone separates the cavity of the middle ear from the dura and brain. It is easy to conceive that a slight degree of caries in this bone will open a way for the entrance of micro-organisms, and their development will lead to either purulent meningitis or to the formation of an abscess. Such an abscess may develop in the temporal lobe of the brain or in the lateral lobe of the cerebellum. Fig. 226 and Plate XXII. show such abscesses.

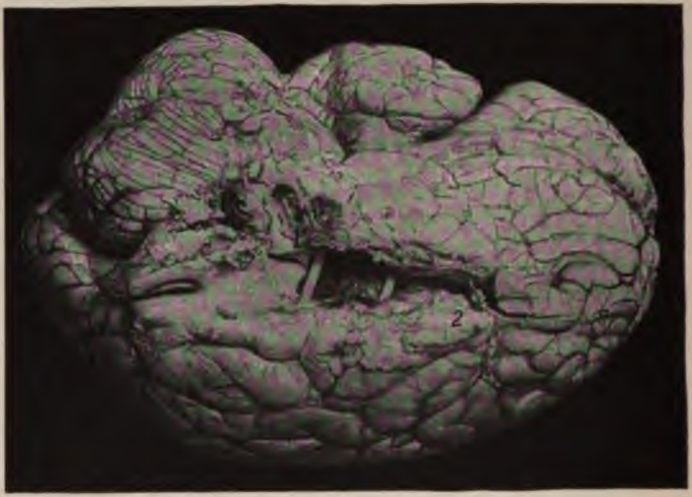
The first attack of otitis media is rarely followed by a brain abscess. It is in the chronic cases, where as time has gone on the bone has been slowly eroded, that a sudden attack of inflammation of the ear goes on to the formation of a brain abscess.

Many cases in adult life are traced to infectious otitis media occurring in childhood, the frequency of this complication of scarlatina and measles being well known.

In all such cases there is a history of a chronic purulent discharge from the ear, which from time to time ceases, but recurs, and then suddenly an acute attack of pain in the ear occurs, which is soon followed by the cerebral symptoms of abscess. In other cases the acute attack of otitis subsides, but the patient does not feel quite well, and after some weeks the signs of a brain abscess occur.

Korner has shown that such abscesses are more common on the right side. Barr in a collection of 76 such abscesses found 55 in the temporal lobe, 13 in the cerebellum, 2 in the pons, and 1 in the crus. Poulsen collected 13 cases of abscess, of which 9 were in the temporal lobe and 4 in the cerebellum.

FIG. 226.



1. Primary abscess cavity connected with sinus in tympanic root. 2. Secondary abscess cavity in temporo-sphenoidal lobe. Both abscess cavities found free from pus at autopsy. (Bacon.)

A third cause of brain abscess is a carious process starting in the nasal cavity or orbit, or an infection after some operation upon these parts. Thus, some years ago, before the importance of aseptic surgery was appreciated, I saw a case of abscess of the frontal lobe, with sudden death, which followed within a week of an operation for the removal of an exostosis of the septum. I have known of several similar cases, and Kahnt and Dreyfuss have collected a number.

Abscess of the brain has been known to follow erysipelas of the face and head.

Metastatic abscess in the brain after abscess and gangrene of the lung is not very common, but occurs. Nähter has collected 8 such cases in a study of 100 cases of pulmonary gangrene. It may also follow bronchiectatic abscess, empyema and pyæmia, ulcerative endocarditis, and abscess in any organ. In these cases there are small multiple abscesses in the brain, whose existence is usually overlooked, as the general symptoms of the original disease obscure the cerebral symptoms.

It has been supposed that in some cases of unknown origin the cerebral abscess is the result of some infection, and many forms of infectious disease—typhus, typhoid, cerebro-spinal meningitis, measles, influenza, and tuberculosis have been considered the starting point of cerebral abscess in various reported cases.

Pathology. Abscess in the brain is the result of a purulent encephalitis. Streptococcus pyogenes, staphylococcus pyogenes aureus, albus and citreus, and pneumococcus have been found in such abscesses. It may present several different appearances. There may be a small, localized, softened condition of the brain tissue, in which on microscopic examination leucocytes, pus cells, and micro-organisms are found. This is the condition in metastatic abscesses. Von Bergman reports a case in which 100 such little collections of pus were found in one brain in a patient who had pyæmia after gangrene of the leg.

There may be a collection of pus within an irregular cavity without distinct wall, but surrounded by a more or less pulpy, broken-down, and hemorrhagic area of brain tissue. The pus is green or brown and fetid. Such abscesses extend rapidly and lead to a fatal termination. Even if evacuated and drained they are likely to go on, and very often set up a meningitis.

There may be a collection of pus enclosed in a thick connective-tissue capsule lying in the white matter of the brain, and quite distinct from the brain tissue. This form occasionally shows a tendency to rapid progress, but is usually of slow growth. It usually lies in or on the brain like a tumor, causing symptoms of pressure; or it may cause no symptoms whatever, and surprise the pathologist at the autopsy. This form is probably a terminal result of the second form described, nature forming a protecting capsule to the disintegrated tissue and shutting up the pus in the thick wall; but such a process may go on with some rapidity, as I have seen a thick wall in a case of abscess of but three weeks' duration. The existence of the wall does not preclude the possibility of an extension of the abscess, and in such cases an infiltration of the brain tissue about it with pus, leucocytes and bacteria has been found. The size of such an abscess varies. It is usually about as large as a walnut, but may be much larger. The rupture of such a capsule may cause sudden death, as in one of my patients. It may rupture inward into the ventricle or outward on to the surface. It is not possible for an abscess to be absorbed.

In many cases of abscess there occurs a complicating meningitis or thrombosis of the cerebral sinuses just prior to death.

Symptoms. The general symptoms of cerebral abscess are, in the order of their importance: 1. Headache, general but sometimes located over the seat of the abscess. 2. A change in mental characteristics, of the nature of irritability, alternating with dulness, imperfect attention, slowness of thought, and defects of memory—a semicomatose condition and appearance of illness out of proportion to the other symptoms. 3. Abnormal temperature. 4. Tenderness of the head to percussion, with change in the percussion note over the seat of the abscess. 5. Facial palsy of the peripheral type upon the side affected. 6. Optic neuritis. 7. General febrile symptoms with occasional chills. The development of these symptoms in a patient who had had an injury of the head, or is the subject of chronic otitis media, or who has been exposed to any of the causes already mentioned, should awaken grave anxiety.

It will be noticed that none of these symptoms are distinctly diagnostic of brain abscess, for all of them may occur in meningitis or in thrombosis of the lateral sinus, and all of them may vary in severity in any given case. I have seen such a combination of symptoms in patients with otitis media purulenta who, by proper surgical treatment, have gone on to recovery without any subsequent evidences of brain abscess.

Thus in two of the cases of traumatic abscess of the brain already mentioned headache was not present. In a case of abscess of the cerebellum which I saw with Gorham Bacon,[1] headache was not a very marked symptom until late in the disease, some time after the beginning of the abscess; and in a case of abscess subsequent to ear disease, seen with McBurney, headache, though present, was never very severe. In other patients, however, I have known it to be intense, so that it formed the chief complaint. It is evident, therefore, that from the degree of headache no conclusion can be arrived at that is of diagnostic value. It is usually stated that the headache in meningitis is more severe and constant than it is in abscess; but if all degrees of headache may occur in abscess, it is evident that this symptom cannot be relied upon in diagnosis.

It is equally difficult to estimate the real importance of mental symptoms. In the first place, the mental capacity and characteristics of different individuals are so different that it is necessary to have known the person prior to his illness in order to reach any definite judgment. A condition of excitement and talkativeness in a person who is ordinarily reserved or stolid is of much more importance than in an excitable and loquacious individual; and, *vice versa*, a condition of apathy and stupor in a man of bright, active intelligence is of much greater diagnostic value than in a dull or stupid person. Anyone who watches a large class going through gymnastic movements under the leadership of an instructor will notice that a certain proportion are quite perceptibly behind the others in their motions, and even in a class of fifty it is rare to find any two who are moving exactly in time, especially when the exercise performed is a novel one. This fact illustrates the variability of the personal equation—the different rate that obtains in the thinking process of different individuals—and unless this fact is kept in mind in judging of the mental state, of the slowness of thought, or of the defects of attention in a person who is ill, very erroneous conclusions may be reached regarding their significance. The effect of illness upon mental processes is not always appreciated, and varies much with the individual, some persons becoming restless, irritable, and more active mentally when ill, others becoming lethargic, morose, and apparently dull. Inasmuch as these variations are observed in all forms of illness, it is obviously erroneous to attach much differential diagnostic value to the mental state in distinguishing between three conditions—abscess, meningitis, and sinus thrombosis—so closely allied.

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[1] American Journal of the Medical Sciences, August, 1895.

The course of the temperature in abscess of the brain is quite variable. The statement has been made by MacEwen,[1] and seems to be borne out by his cases, that it is common in abscess of the brain to find a persistently low temperature with little variation. MacEwen states that during the preliminary period the temperature has been slightly above normal. During the period of full development of the abscess the temperature is about normal or slightly subnormal, from 97° to 99° F.; and that in the terminal stage if the abscess bursts the temperature rises within a few hours with a bound to 104° or 105° F., but if the abscess is evacuated by operation the temperature rises to 100½° or 101° F., and in a few hours comes down to below 100° F., remaining near the normal until recovery ensues. He contrasts sharply this run of temperature with that found in infective thrombosis of the lateral or cavernous sinuses, in which very high temperatures with very great remissions, resulting in a most irregular curve between 106° and 97½° F., have been observed. He also contrasts it with the temperature curve of meningitis, in which the temperature is high, between 103° and 104° F. constantly, without the great remissions occurring in thrombosis. Okada found a marked rise of temperature and a febrile course in 46 of 88 cases of abscess of the cerebellum; in 15 the temperature was normal; in 15 it was subnormal; in 8 there was a rise of temperature only at the onset, in 4 only at the very end. A study of the temperatures occurring in three successive cases of cerebral abscess shows that any conclusion from a study of temperature is not reliable. Thus in a case seen with Bacon,[2] the curve was one which suggested thrombosis of the sinuses, there being very high temperatures, followed by sudden remissions two or three times a day for several days prior to the operation. In this case the abscess was successfully evacuated and the patient recovered.

In a second case the temperature chart corresponded pretty nearly to that described by MacEwen; the temperature was not above 98½° F. or below 98° F. during the week of illness.

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[1] Pyogenic Infective Diseases of the Brain. MacMillan Co., London, 1893.

[2] New York Medical Journal, August 15, 1896.

In a third case the temperature chart was much more suggestive of meningitis, varying from 101° to 104° F. during the entire illness of eight days. Hence the conclusion must be reached that the course of temperature is not to be relied upon as a diagnostic sign.

Sometimes the rapid emaciation of the patient, his cachectic appearance, with yellow skin and general evidences of a septic state, are so marked as to suggest a latent abscess, even when the temperature is low.

MacEwen was the first to call attention to the difference in the percussion note over the side of the head upon which the abscess lies. It is somewhat difficult to elicit this physical sign. The stethoscope is to be placed upon the forehead in the median line, or upon some bald spot upon the head, as any contact with the hair vitiates the test. The head is to be struck with a percussion hammer tipped with rubber. The note elicited varies very much in different skulls, it being very dull and flat in a baby, where the bones are very thin, and of higher pitch and clearer in the adult. The patient's head should not rest upon the pillow while the test is applied. MacEwen believes that as the intracranial pressure increases the percussion note becomes higher in pitch and of greater resonance, and that this resonance increases as the disease advances. In four cases of brain tumor I have been able to confirm this statement, the note elicited being of greater resonance and higher pitch over the side of the tumor than upon the opposite side. In all these cases there was a great distention of the lateral ventricles with fluid, a condition which MacEwen also mentions as affecting the percussion note. In two other cases of brain tumor, and in two cases of cerebral abscess, no essential difference could be determined between the two sides. It is evident, therefore, that this sign is one which requires further study before it can be said to be diagnostic, though it appears to be of considerable value, and should be more widely known and more commonly noticed.

Tenderness to percussion or to pressure is a symptom emphasized by Horsley as occurring in brain tumors, and it has been observed in brain abscess. It appears to depend in both cases upon the nearness of the tumor or abscess to the surface or upon a thinning of the cranial bones in the vicinity of the tumor or abscess. Such a thinning of the cranial bones occurs in some cases in a very remarkable manner. Thus in a patient suffering from tumor of the brain, seen at the New York Hospital with Peabody, the symptoms pointed to a tumor located in the posterior central convolution at the junction of its upper and middle thirds, there being a paralysis with anæsthesia of arm and leg of the opposite side, and over this region there was great tenderness to percussion and to pressure and a marked difference in the percussion note. At the autopsy in this case the bone was so much reduced in thickness over this region as to be translucent, affording a sharp contrast to the skull in other parts. The tumor, however, did not lie in contact with the skull nor upon the cortex, but was at least one inch below the level of the cortex, within the superior parietal lobule and nearer to the falx than to the dura of the convexity.

Facial palsy is a symptom which has been observed in abscess of the cerebellum from pressure upon the pons at the exit of the seventh nerve. It is not to be forgotten, however, that in ear disease the facial nerve is frequently affected in its passage through the middle ear, and therefore this intracranial symptom must not be overestimated as indicating either meningitis or abscess.

Optic neuritis is a sign of great value in the diagnosis of cerebral abscess and especially in its differentiation from meningitis. In Okada's cases two-thirds of the patients developed optic neuritis in one or both eyes. It often occurs in thrombosis of the lateral sinus, and therefore is of less value in differentiating abscess from this condition. It is to be remembered, however, that many abscesses of the brain run their course without the development of this sign.

It seems evident from these considerations that in the diagnosis of cerebral abscess very little reliance is to be placed upon any one symptom. It is the combination of these symptoms, however, and a certain order of their development, which makes the diagnosis clear. It is possible to divide the course of the disease into three distinct periods. These are not sharply separated from one another, but in looking back over a case it is quite evident that they are distinct. The history of the following case will illustrate the course of the disease following otitis media:

Male, aged thirty-six years. Had had an attack of scarlet fever at the age of fifteen years, which was attended by an inflammation of the middle ear, leaving a chronic discharge from the right ear which had varied in intensity and severity from time to time during twenty years. This otitis media had not produced any pain and was not attended with any degree of deafness. Occasionally the discharge increased considerably and then the ear required some treatment. In March he caught cold, suffered from considerable pain in the ear and from a sudden increase in the discharge of pus. The onset of this acute attack was attended by a rise of temperature, which lasted three or four days and then subsided. The discharge was quite profuse and was treated by antiseptic washings. It lasted about one month and then gradually dried up, so that the specialist under whose care he was discharged him cured of the otitis media, the last week in April. This attack had differed in no respect from numerous preceding attacks, and was not considered of any particular importance by himself or his family, but during this month of treatment he suffered from a constant headache over the right side of the head, the side upon which the ear was affected; and although this headache was only occasionally intense, yet he was never free from it entirely. During the month he became more and more nervous and irritable, also unduly apprehensive as to his own condition, and at times he acted in a markedly hysterical manner. He also became gradually more and more depressed and somewhat dull, and on two occasions during the month he vomited suddenly without any nausea or digestive cause. He also suffered occasionally from dizziness, and on several occasions felt faint when the ear was being treated.

On April 29th, a few days after the discharge from the ear had ceased, his headache suddenly became intense and he vomited twice. He became very much disturbed in his mind, was nervous, apprehensive and irritable, felt faint and dizzy. His temperature was 98° F. and his pulse 60. His tongue was coated thickly, his breath was foul, and he had the appearance of a man suffering from some septic condition, but there was no discharge from the ear, there was no tenderness about the mastoid, there was no tenderness of the head to percussion, the optic disks were clear, the pupils reacted promptly and were equal, the knee-jerks were normal, there was no disturbance of his gait, and although he was complaining he was able to be up and about. This condition continued on April 30th and on May 1st, but on May 2d, after brisk purgatives had acted he seemed to be better, though he was still apprehensive and still suffering from headache, nausea, and somnolence. His temperature had remained constantly at 98° F. and his pulse had varied from 60 to 80. The only new symptom which had appeared was a dilatation of the right pupil.

On May 4th his condition had changed markedly for the worse. He had suffered intensely from headache, the somnolence had increased, and he had become more dull and was aroused with some difficulty, and when aroused was fretful and irritable, and did not talk spontaneously, but only in response to questions. This somnolent condition alternated with a state of great restlessness, in which he turned constantly from side to side, complaining greatly of general discomfort and of pain in the right side of the head. His right pupil was still dilated, and though both pupils reacted to light, the optic disk of the right eye was distinctly congested and slightly swollen; but careful examination failed to reveal any paralysis, any change in the reflex activity, any disturbance of sensation, and there was still no tenderness over the mastoid and no tenderness to percussion of the head. The temperature was still 98° F. and the pulse 52 to 60, and occasionally irregular. At this time a diagnosis of abscess of the brain was made, and an operation was advised and agreed to. It was thought best, however, to defer it twenty-four hours. On May 5th the general condition had become somewhat worse. The temperature was still 98° F., the pulse 52, but varying much in its regularity, though never intermittent. His respirations were regular. He lay in a state of stupor, was aroused with great difficulty, his right pupil was still dilated, the optic disks congested, and the left side of the face was slightly flattened, this being the only evidence of paralysis that could be elicited. The knee-jerks were still normal and equal. He was restless in spite of his stupor, and occasionally had an expression of pain upon his face and would put his hand to the right side of his head as if in pain. In spite of the absence of more definite symptoms it was decided to make an exploratory trephining over the right temporo-sphenoidal lobe, and preparations were begun for this operation; but before the operation was begun he suddenly had a spasm of the entire left side of the body, and died. No autopsy was permitted, but in view of the sudden death it seemed probable that an abscess of the brain which had developed subsequent to the ear disease had suddenly ruptured, probably into the lateral ventricle.

In reviewing the case it is evident that the first period of the disease coincided with the month during which the patient was being treated for otitis media, and during which he had suffered from headache and irregular cerebral symptoms. The second period of the disease, or the period of acute onset, began on April 29th and lasted for three days, merging gradually into the third period of termination, the beginning of which was shown by the onset of stupor.

A reference to the published histories of abscesses by Bacon[1] and others will show that three periods of similar limit can be distinguished, and therefore it seems evident that in the diagnosis of abscess of the brain the combination of the symptoms in a regular succession of stages rather than the existence of any one symptom is of the greatest aid in diagnosis.

The distinguishing characteristics of these periods may be briefly enumerated as follows: The preliminary period, during which slight, irregular, and variable cerebral symptoms, chiefly paroxysmal pain and a dull ache in the head, occasional vomiting, and a lack of power of concentration, with mental fatigue developing easily and unusually, are the chief symptoms. During this period occasional rises of temperature with chilly sensations or even an occasional chill may be noticed, and the patient has the malaise of a slight septic infection. During this period the ear may discharge freely or the discharge may gradually cease. A sudden cessation of the discharge, and a sudden fall of temperature to a point below normal two or three days after the discharge has ceased, is a quite common method of termination of this stage; hence the cessation of a discharge in a chronic otorrhœa, if the discharge has been attended by febrile symptoms for a few weeks or days, and if the cessation is attended by a sudden fall of temperature, the patient continuing to feel ill, with indefinite cerebral sensations, is very suggestive of the formation of cerebral abscess.

In the second stage all the symptoms that have been already enumerated may occur in varying combination and in varying severity: the increasing mental stupor, alternating with irritability and restlessness; the general septic appearance of the patient, either with irregular temperature or low temperature and with persistently slow pulse; more serious disturbance of digestion than can be accounted for by the apparent gastric conditions, and the development of objective cerebral symptoms; difference in the size of the pupils; optic neuritis, facial palsy, increase of reflexes in the limbs of the side opposite to the lesion—all these symptoms are suggestive of cerebral abscess. It is in this stage that operation should be performed if the diagnosis seems reasonably probable, for if the patient goes on to the terminal state, in which deep stupor is the chief characteristic, the operation will probably be too late.

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[1] Handbook of Otology, 3d ed. Lea Bros. & Co., Philadelphia, 1900.

The position of the injury in such cases will indicate the point to be trephined. In abscesses of the convexity of the hemisphere following trauma the local symptoms of lesion will be those already studied as characteristic of cortical lesion, especially unilateral spasm and paralysis or aphasia, hemianopsia, or hemianæsthesia.

There are very few localizing symptoms of cerebral disease in abscesses following otitis media, for the functions of the temporo-sphenoidal lobe, and especially of that part of it which lies next to the petrous portion of the temporal bone, and the functions of the cerebellum, so far as its lateral lobes are concerned, are not fully known. There are apparently few distinct cerebral symptoms that develop uniformly in abscess of these regions; but in abscess of the temporo-sphenoidal lobe of the left hemisphere a certain form of aphasia has recently been observed which deserves attention. It will be remembered that in the first and second temporal convolutions are located the memories of words heard, the relative degree of development of this function of the cortex varying much in individuals of different degrees of education. It is also to be remembered that in the occipital lobe are located the memories of objects and of words seen. In 1889 Freund[1] described a condition which he named optical aphasia. This consists in an inability to name objects which are seen and recognized and whose use can be described. Sometimes the name can be recollected if other senses—touch, smell, and taste—are called in to aid sight. Freund says: "Granting that the function of naming objects seen—*i. e.*, making an association between the visual pictures of the object and the auditory picture—is performed through the association tracts between the occipital lobe and the speech centres, the explanation of a disturbance of this function is found in a lesion which interferes with the action of the association tract. Such a lesion in right-handed persons must lie in the temporal lobe of the left hemisphere." This localization has been substantiated by Freund by several autopsies. Arnold Pick[2] in a well-observed case of cerebral abscess has described this symptom of optical aphasia fully and made it the basis of a diagnosis of the localization of a cerebral abscess which the operation confirmed. In a case reported by Bacon[3] I had the opportunity of studying this defect of speech. It is alluded to in Bacon's history as verbal amnesia, or an aphasia of conduction. I was unaware of the description of Freund at the time, and the case was seen before the appearance of Pick's article. I can confirm fully the description of Freund and of Pick, and therefore call attention of English-speaking physicians to this symptom. When the patient is shown objects he knows them, but he cannot name them. He can understand and recognize their names, however. He can talk well and will describe the object, avoiding its name. Thus he will say, when shown a knife, "Yes, I know it; it is used to cut with," etc. He can repeat the name perfectly. He can often name the object if he handles it or smells it or tastes it, but the name does not come to his mind when he sees the object. I noticed in Bacon's case a further difficulty which this patient, who was very intelligent, also recognized. When the name of an object was given it was not possible for him at once or easily to call to his mind the appearance of the object. In a word, it was as difficult for him to make an association between his auditory and his visual centres as it was to make one between his visual and his auditory centres. This is a feature of the condition not noted by Pick. This symptom of optical aphasia, or intercortical sensory aphasia, as I name it, is due to a suspension of function of an association tract lying in the temporo-occipital lobes and occupying the white matter deep under the cortex. It lies in a position in which it is almost inevitably affected by abscess of the temporal lobe developing after otitis media. This symptom should therefore always be looked for, and may be regarded of highest value in locating the abscess. In left-handed persons it will occur in abscess of the right side. (See Plate XIX. and page 466 on aphasia.)

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[1] Arch. f. Psych., xx., 276.

[2] Praguer med. Woch., 1896, xxi., 5.

[3] New York Medical Journal, August 15, 1896.

When an abscess in the temporal lobe extends inward it may compress the internal capsule and thus give rise to symptoms of slowly increasing hemiplegia, hemianæsthesia, and hemianopsia upon the opposite side of the body. In a case of Oppenheim's, operated upon by Jansen, these symptoms were present. There was also a conjugate deviation of the eyes to the side of the abscess.

The chief local symptoms of cerebellar abscess are vertigo and a staggering gait. The vertigo is sometimes so severe that the patient cannot raise the head from the pillow, and cannot even attempt to stand. It is often associated with vomiting and sometimes with double vision and nystagmus. The vertigo may lead to a staggering gait, but this may develop without true vertigo. The patient walks like a drunken man, grasps at objects for support, and is unable to walk a line. Sometimes there is a marked tendency to stagger toward one side. This is usually toward the side of the abscess. In cases of cerebellar abscess the pressure exerted upon the side of the pons or medulla may lead to symptoms of cranial nerve involvement upon the side of the abscess. In cerebellar lesions there is sometimes a loss of knee-jerk on the side of the lesion. If there is at the same time a pressure on the pons there will be an exaggeration of knee-jerk on the other side. The absence of symptoms of temporal lobe lesion in a case where abscess is probable may lead to an exploration of the cerebellum.

Diagnosis. The diagnosis of cerebral abscess does not appear to be very difficult in ordinary traumatic cases, for there is a history of the original injury, of its exact location, and of the development of a series of cerebral symptoms pointing to a localized disease in the brain. If, then, the location of the wound in the head and the localization of the cerebral disease based upon the nervous symptoms coincide, there is little doubt as to the existence of the abscess.

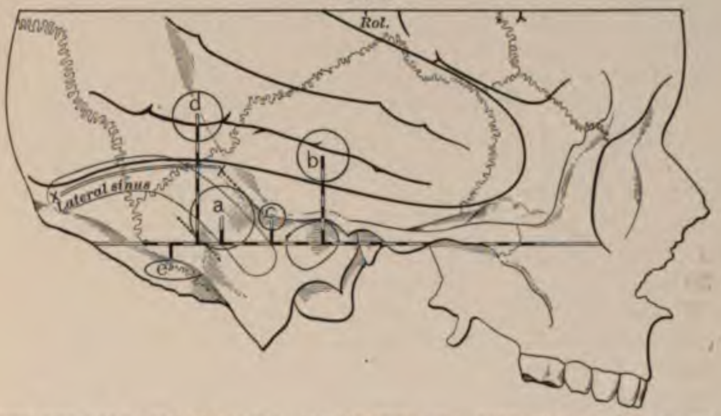
Where there is no history of a cause the diagnosis is much more difficult, as it is sometimes impossible to differentiate the condition from acute encephalitis. Thus in a patient seen with James the development of acute cerebral symptoms during ten days suggested abscess. The patient was a pregnant woman, in the eighth month. No history of any of the causes of abscess could be obtained. She developed exophthalmus of the right eye and a slowly increasing left hemiplegia, with high temperature. The autopsy showed a large abscess on the surface of the right frontal region, but no source of infection could be ascertained.

In cases of otitis media three separate conditions may supervene which are often difficult of differentiation. These are cerebral abscess, meningitis, and thrombosis of the lateral sinus. The relative frequency of these conditions is about the same. Thus in thirty-six cases, collected by Poulsen, of cerebral complications of ear disease there were thirteen cases of abscess, twelve cases of thrombosis, and eleven cases of meningitis.

In meningitis there is usually a more rapid onset and progress of the symptoms than in brain abscess. In meningitis the headache is associated with hyperæsthesia to sound and to light, and to touch all over the body, symptoms which are absent in abscess. In meningitis the temperature is high, varies between 101° and 104° F., and rarely if ever goes below normal. It does not show the rapid rises and falls which occur in thrombosis of the sinuses. In meningitis the pulse is rapid, irregular, and intermittent, while in abscess it is slow. In meningitis there are occasional twitchings or spasm of the limbs, or convulsions; strabismus appears early, trismus is common, and pain and rigidity of the neck are complained of, as the disease advances. These symptoms are wanting in brain abscess. In case of doubt lumbar puncture will reveal the presence of micro-organisms in the cerebro-spinal fluid in meningitis, but not in brain abscess.

In thrombosis of the lateral sinus high fever with pyæmic variations in range and frequent chills are the rule. The temperature in the course of twenty-four hours may twice sink below normal and rise to 105° F. The pulse is very rapid and irregular, but not intermittent. There is tenderness, swelling, and œdema over the mastoid process, and œdema of the neck, and the jugular vein may stand out as a hard blue cord on the side of the neck. Exophthalmus on one side, and even swelling of the conjunctiva, may be seen. There is often a marked venous congestion of the scalp. Choked disk appears

FIG. 227.



Lateral aspect of a small adult skull. The illustration shows the relations of the lateral sinus to the outer wall of the cranial cavity and the position of the trephine opening (a), which should be made when it is deemed necessary to expose it. The base line (Reid's) passes through the middle of the external auditory meatus, and touches the lower margin of the orbit; it is marked out in eighths of an inch, as are also the perpendicular lines drawn from it. The measurements are made along the base line from the middle of the bony meatus. The drawing also shows the convolutions of the temporo-sphenoidal lobe, the Sylvian fissure, and the position of the lower end of the fissure of Rolando (*Rol.*) x x indicates the site of the tentorium as far as it is in relation to the external boundary of the skull. The anterior x shows the point where the tentorium leaves the side of the skull and is attached to the superior border of the petrous bone. a, trephine opening to expose sinus, five-eighths of an inch in diameter, its centre being one inch behind and a quarter of an inch above the middle of the bony meatus. This opening can easily be enlarged upward and backward and downward and forward (see the dotted lines) by suitable angular cutting bone forceps. It is always well to extend it forward, so as to open up the mastoid antrum (c) and the gutter of the carious bone (if there be one), which leads from the antrum, tympanum, or meatus down to the bony groove. The position of the trephine openings, which must be made for the relief of inflammatory intracranial affections, secondary to disease of the ear, other than for sinus pyæmia, have been added to the drawing for the sake of contrast and completeness. They are as follows: b, trephine opening to explore the anterior surface of the petrous bone, the roof of the tympanum, and the petro-squamous fissure, half an inch in diameter, its centre being situated a short inch (seven-eighths of an inch) vertically above the middle of the meatus. At the lower margin of this trephine hole a probe can be insinuated between the dura and bone, and made to search the whole of the anterior surface of the petrous. c, trephine opening for exposing the mastoid antrum, a quarter of an inch in diameter, and half an inch behind and a quarter of an inch above the centre of the meatus; or a quarter of an inch above the centre of the meatus and a quarter of an inch behind its posterior border. The trephine should be directed inward and slightly downward and forward. When a superficial disk of bone has been removed, it is well to complete the operation with the gouge. A larger trephine may with advantage be employed, especially in adults d, trephine opening for temporo-sphenoidal abscess, half an inch in diameter. Situation recommended by Barker, one inch and a quarter behind and one inch and a quarter above centre of meatus. The needle of the aspirator is to be directed at first inward and a little downward and forward. Birmingham prefers one and three-fourths of an inch above, in order to avoid the lateral sinus. e, trephine opening for cerebellar abscess, half an inch in diameter and one inch and a half behind, and a quarter of an inch below the centre of the meatus. Birmingham prefers two inches behind and one inch below to avoid the occipital artery. The anterior border of the trephine should just be under cover of the posterior border of the mastoid process. The drawing shows that a trephine hole made in this situation is far away from the lateral sinus, and that the trocar and canula of the aspirator, if directed forward, inward, and upward, would hit an abscess occupying the anterior part of the lateral lobe of the cerebellum, which is the usual site of collections of pus in this part of the brain.—From *Brain Surgery*, by Starr, p. 193. (Ballance.)

early in the course of the case. All of these symptoms serve to distinguish the disease from abscess.

The diagnosis of cerebral abscess arising under other conditions is never positive. But when a cause is present and cerebral symptoms such as have been described occur in a definite series of stages the probability of an abscess is very great.

The **prognosis** will depend wholly upon the possibility of evacuating the abscess and draining it successfully.

Treatment. The only treatment in a brain abscess is surgical. In a collection of 55 cases of abscess of the brain which have been operated upon I found that 28 occurred after injuries, 24 after ear disease, and 3 after typhoid fever. Of these 34 recovered and 21 died. Oppenheim has recently published more extensive statistics. In 60 cases of traumatic abscess operated upon 38 cases recovered. In 196 cases of otitic abscess 96 were cured. In cases of injury and fracture of the skull trephining should be undertaken early, the local trauma combining with the local symptoms to guide the surgeon.

Fig. 227 shows the relation of the skull to the convolutions of the temporal lobe and cerebellum, and also the position of the lateral sinus. The rules given by Ballance for trephining in the cases developing after otitis media are given in the explanation of the figure.

While the differentiation of abscess of the brain from thrombosis of the lateral sinus and from meningitis is of great interest to the physician, yet it must be clearly stated that it is not absolutely necessary to differentiate these conditions before deciding upon the question of surgical treatment. It must be agreed that all three conditions are necessarily fatal to the patient, and, therefore, that any means which may offer a reasonable amount of relief to any or all should be employed at the earliest possible moment without waiting for absolute diagnosis. If the condition is one of meningitis the patient will die whether operated upon or not, and the operation will not do any harm. If the condition is one of abscess or of sinus thrombosis, the operation may give relief, and if one condition is not found the other may be sought for. When it is considered that a few years ago both these conditions were considered necessarily fatal, and when it is known that within the last two years the number of successful operations reported for brain abscess has been large, it is evident that surgeons are appreciating the necessity of this procedure. And when it is further learned that of the 92 abscesses reported to July 1, 1895, by Körner 51 recovered, and of the 79 cases of sinus thrombosis 41 recovered, it is evident that surgery has done a great work in the treatment of these conditions. This is not the place to give detailed histories or analyses of these operations. It is evident, however, from their study, that the cases in which success did not attend surgical procedure were those in which the operation had been delayed too long or had not been thoroughly exploratory. When it is considered that the part of the brain which is usually involved in the abscess, viz., the temporal lobe or the cerebellar hemisphere, may be destroyed without producing any local symptoms, and when it is found that the patients, after the evacuation of abscesses as large as a hen's egg, may recover without the apparent loss of any cerebral function, it seems justifiable at the time of the operation to explore the brain thoroughly in these regions without fear. Large incisions may be made into the base of the temporo-sphenoidal lobe or into the lateral lobe of the cerebellum without doing any harm except from the attendant hemorrhage. If the abscess is not found on exploring the temporal lobe it is advisable to explore the cerebellum.

Inasmuch as almost all brain abscesses have a thick capsule and contain very thick pus, it is not sufficient in the exploration to merely puncture with a needle or with a small trocar, as the wall may be pushed before these instruments and not punctured. I would strongly urge the use of an instrument devised, I believe, by Horsley for the exploration of the deeper parts of the brain. This instrument resembles closely a large ear speculum, but is sharp at the end and is placed upon a scissors-like handle and is split in two parts, so that after its introduction its sides may be separated, allowing a view of the bottom of the cavity into which it is introduced. By pushing such a speculum into the brain tissues gently an incision of the vessels can be avoided, the resistance of the capsule of the abscess more easily detected, and the wall can be seen before and during the time of its incision.

The question of drainage of these abscesses is also one of great interest. It is to be remembered that the cavity of the abscess in the majority of cases is large and that it will require some time for it to contract or for its walls to approximate, and for the cavity to be obliterated. Unless thorough drainage is maintained during that time the abscess will reappear. The tendency of the brain tissue to collapse is so great that unless the drainage is maintained by a fairly stiff tube it will not be successful; hence it has seemed to me that a small roll of rubber tissue or a decalcified bone tube is preferable to horse-hair or to gauze.

The practical conclusion, therefore, which experience has taught is this: that when a cerebral abscess seems probable from the history of the case and from the symptoms which have developed, and the general progress of the case demonstrates the existence of an increasing and serious focal disease of the brain, it is advisable to operate, even though the symptoms may not be absolutely typical and may present many variations from their usual form.

The rapidly increasing percentage of recoveries after operation warrants a degree of boldness, provided the surgeon is careful in the observance of every rule of asepsis, without which precaution such a venturesome operation would be criminal.

CHAPTER XXXI.

THROMBOSIS OF THE VENOUS SINUSES.

THE various venous sinuses of the cranium and their communications are shown in Figs. 207 and 228. A thrombus may form in any of these sinuses, but it is in the lateral or transverse sinus that it is most commonly found.

Etiology. Thrombosis is the result of a phlebitis of the wall of the sinus due to an extension to it of an inflammatory process, usually of a septic nature, in the immediate vicinity, viz., from the dura mater in pachymeningitis, from the bone and veins after otitis media, or chronic nasal disease or orbital disease. Occasionally thrombosis may be secondary to septic processes elsewhere, especially abscesses and carbuncles. In all these cases the thrombus is infected, contains bacteria, and if it breaks up and its particles are carried onward it causes infective emboli in other organs, and pyæmia. In children dying of marasmus, in chronic invalids who have suffered from exhausting diseases and have died of heart failure, in a few cases of fatal infectious fevers, and in cases of chlorosis, thrombi have been found in the sinuses without phlebitis, and in these cases they have been ascribed to the state of the blood. The pressure of a brain tumor or of a dural tumor occasionally leads to thrombosis. In a collection of cases by Allport[1] 118 out of 128 cases were secondary to otitis media. The majority of cases occur between the ages of fifteen and twenty-five years.

Pathology. The thrombus may be confined to one sinus or even to a portion of it, or it may invade several of the sinuses by gradual extension. It often projects into the veins which lead out of the sinus. It is usually a soft clot adherent to the wall of the sinus. The wall is slightly roughened. If time has permitted, the clot may become hard or even organized. If septic it is foul in odor, broken down, yellowish-green, and purulent, and the adjacent wall is covered with pus. Thus in a patient, operated upon for me by Abbe, with lateral sinus thrombosis secondary to ear disease, a purulent, fetid, green mass occupied the sinus near the ear, a long clot was extracted from the posterior part of the sinus, another was pulled up from out of the jugular vein, in which a hard thrombus had formed which could be felt in the neck; the wall near the purulent mass was rough, softened, evidently infiltrated with pus. In this case the jugular was tied before the sinus was opened. The patient recovered.

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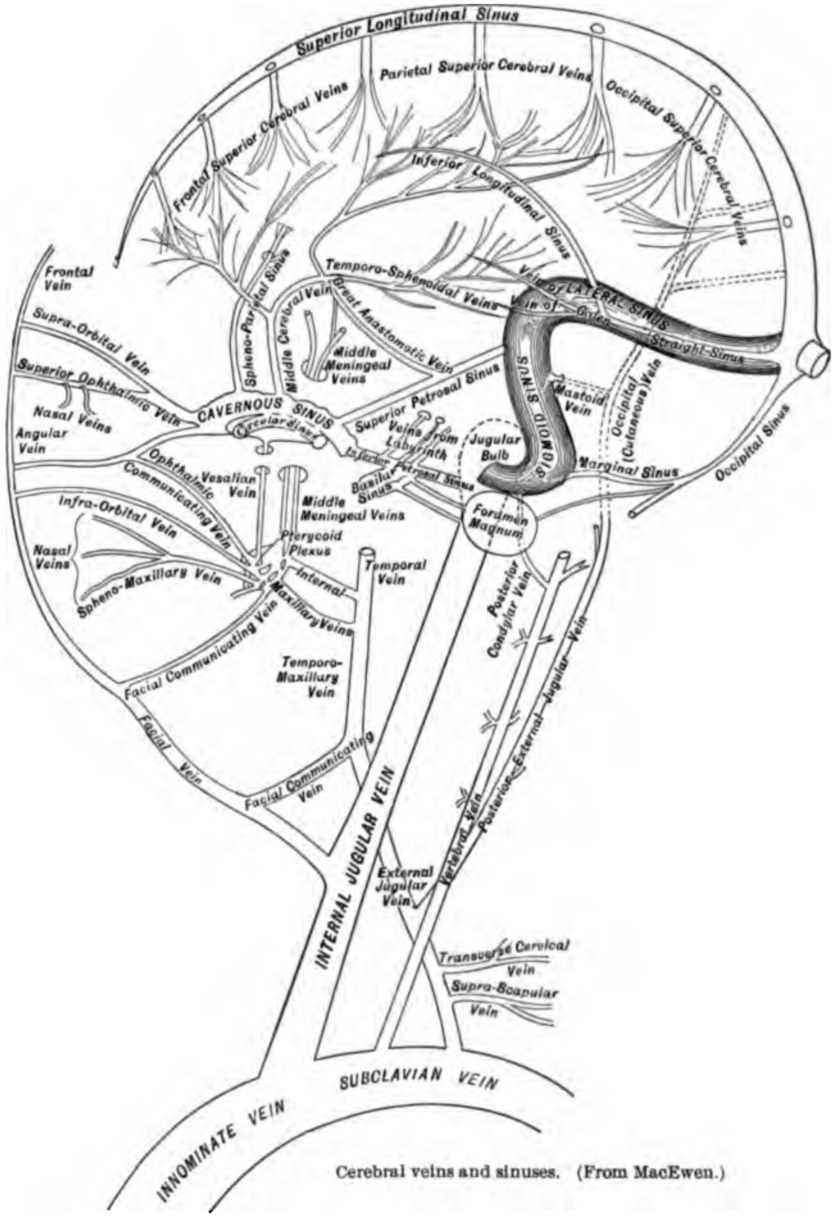
[1] Journal of the American Medical Association, 1902, p. 690.

In patients who die a congestion of the brain is found in the area whose blood empties into the sinus that is the site of thrombosis,

FIG. 228.



Cerebral veins and sinuses. (From MacEwen.)

and an acute hemorrhagic softening with infarction has been found. In these cases also multiple septic emboli are found in other organs.

Symptoms. Thrombosis of the longitudinal sinus is extremely rare and occurs chiefly as the result of septic pachymeningitis or in cases of marasmus. In both conditions its occurrence is rarely diagnosticated, the only symptom indicative of it being a marked venous stasis in the scalp. When this is present the veins of the entire cranium are distended, including those of the orbit and eye. In a case of Oppenheim's, due to chlorosis, choked disk was seen in both eyes. Nose-bleed has been observed, and bulging of the fontanelle in infants.

Thrombosis of the lateral sinus is a frequent complication of otitis media, and its diagnosis rests partly on the prior existence of this disease with its symptoms of earache, purulent discharge, headache, and general malaise. It causes a venous congestion of the mastoid region and œdema over the mastoid, with pain and tenderness; a swelling and thrombosis in the jugular vein which can be felt as a hard cord in the neck in one-third of the cases, or an unusual emptiness of this vein detected in inspiration (Gerhardt's symptom), a swelling of the cervical glands, pain in the neck on motion of the head and sometimes on swallowing, nystagmus and occasionally torticollis, the patient holding the head toward the affected side to avoid pain. There is a choked disk in over one-half of the cases within the first week. The general constitutional symptoms of thrombosis of the lateral sinus are very similar to those of brain abscess, and the reader is referred to that chapter for the few points of distinction which can be made. The disease often begins with a chill and high fever (104°–106° F.), headache, vomiting, vertigo, stupor, and delirium, and these are soon followed by local cerebral symptoms, such as hemispasm or paralysis, aphasia, or some affection of the cranial nerves. The fever, as a rule, continues high with frequent intermissions, and, if septic emboli lodge, each gives rise to a chill, a fall of temperature below normal, and a rise to 105° or 106° F. Profuse sweating, rapid, irregular pulse, diarrhœa, jaundice, and local signs in lungs, liver, spleen, and kidneys establish the existence of pyæmia. Death follows in the course of ten days unless surgical treatment is efficacious.

Thrombosis of the cavernous sinus occurs in connection with nasal and orbital disease. Its local symptoms are œdema and swelling, with venous congestion in the face and about the eye, nasal hemorrhage, exophthalmus, and distention of the retinal veins. Various forms of strabismus invariably appear as the oculomotor and abducens nerves pass through the walls of the sinus.

The **prognosis** is absolutely fatal excepting in the cases where an operation can be performed in time—viz., in thrombosis of the lateral sinus.

Treatment. The treatment of thrombosis of the lateral sinus is by trephining the bone over the sinus, or by chiselling away the mastoid process and laying bare the sinus, which is then opened freely and cleaned out. It is always well to tie the jugular vein prior to the opening of the sinus. If a clot is found and taken away free flow of blood must be allowed to wash out the remnants of the thrombus, and the inner wall of the sinus may be scraped with a small curette. The hemorrhage is easily arrested by pressure, and the spontaneous tendency of the sinus wall to heal is remarkable. The only cases which prove fatal are those in which the thrombus is not entirely evacuated, in which secondary thrombi lead to pyæmia, or in which gangrene of the wall of the sinus leads to a secondary thrombosis. In a collection of 150 cases from recent literature 90 cases were found to have recovered from this operation. For full details of the operation the reader is referred to MacEwen's[1] book and to the articles by Koerner[2] and Jansen.[3]

[1] Pyogenic Infective Diseases of the Brain. MacMillan Co., 1893.

[2] Die otitische Erkrankungen des Gehirns, Jena, 1894.

[3] Berliner klin. Wochenschrift, 1891, p. 1160, and 1901, p. 721.

CHAPTER XXXII.

TUMORS OF THE BRAIN.

Tumors of the brain are not uncommon relatively to other nervous diseases, and are seen both in children and in adults. Collections of cases of brain tumors have been published during the past twenty years by Bernhardt,[1] Steffan,[2] Bramwell,[3] Mary Putnam Jacobi,[4] Mills and Lloyd,[5] Knapp,[6] Bruns,[7] Oppenheim,[8] and myself.[9] It is possible by the study of these collections of cases to make a fair estimate of the relative frequency of different varieties of tumor and of the various situations in the brain in which tumors grow, and also to estimate the probabilities of success in finding and removing a tumor. Without attaching too great importance to statistics on this subject, I have selected as the basis of this chapter, from the collections already named, 600 tumors. These are tabulated in Table I., in which tumors in individuals below the age of eighteen years, chiefly children, are separated from those in adults, for the purpose of contrast. In this table no tumors are included which have been removed or in which surgical interference has been attempted. Yet during the past twelve years over 400 tumors have been operated upon.

The percentage of cases open to operation has been variously estimated by different authors, and their results are demonstrated in Table II. The various authors cited have made collections of cases with post-mortem records, and from a study of the condition found at autopsy have estimated the probability of success in the removal of the tumor found. In this table I have included 100 cases under my own care, some without autopsies. In 20 of these the tumor was found either at the operation or at the autopsy to be accessible. From this table it is evident that about 9 per cent. of tumors can be removed.

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[1] Beiträge zur Symptomatologie und Diagnostik der Hirngeschwülste, 1881. Also yearly summary in Virchow's Jahresbericht, 1881 to 1902.

[2] Die Krankheiten des Gehirns im Kindesalter. Gerhardt's Handb. der Kinderkrankheiten, 1880, vol. v.

[3] Intracranial Tumors, 1888. Edinburgh Medical Journal, December, 1894.

[4] Reference Handbook of Medical Sciences, 1888.

[5] Pepper's System of Medicine, vol. v.

[6] Intracranial Growths, 1891.

[7] Encyclopädisches Jahrbuch, 1896.

[8] Nothnagel's allg. Path. u. Therap., 1896.

[9] Medical News, Jan. 12, 1889; Intracranial Tumors, Keating's Cyclopædia of Children's Diseases, 1890. Brain Surgery, 1893. New York Medical Record, February, 1896. Montreal Medical Journal, Nov. 1897. Journ. Nerv. and Ment. Dis., June, 1903.

TABLE I.

(The first column are children's tumors; the second column adult's tumors.)

Situation.	Tuberculous.		Sarcomatous.		Gliomatous.		Glio-sarcomatous.		Cystic.		Carcinomatous.		Gummatous.		Other varieties.		Total.	
I. Cortex cerebri . . .	13	9	1	46	6	19	...	8	...	...	1	19	...	13	...	12	21	127
II. Centrum ovale . . .	6	2	5	7	1	11	1	4	15	...	1	3	1	...	5	4	35	51
III. Cerebral axis:																		
1. Basal ganglia and lateral ventricles . . .	14	3	5	8	3	9	...	...	1	1	1	2	...	1	3	5	27	34
2. Corpora quadrigemina and crura cerebri . .	16	1	3	2	1	2	...	5	...	...	...	1	...	...	1	7	21	14
3. Pons	19	11	5	1	10	...	2	1	1	...	...	2	...	3	1	...	38	17
4. Medulla	2	...	...	...	...	1	...	...	1	...	2	...	...	...	1	...	6	2
5. Base	...	3	1	3	...	2	1	1	1	..	1	...	...	...	4	1	8	9
6. Fourth ventricle . .	1	...	1	1	1	...	...	...	...	...	1	2	...	...	1	1	5	4
IV. Cerebellum	47	8	10	13	15	8	1	6	9	...	3	...	...	...	11	10	96	41
V. Multiple tumors . . .	34	4	3	5	...	2	...	...	2	1	...	2	1	3	3	1	43	57
	152	41	34	86	37	54	5	25	30	2	10	31	2	20	30	41	300	300

TABLE II—PERCENTAGE OF BRAIN TUMORS REMOVABLE.

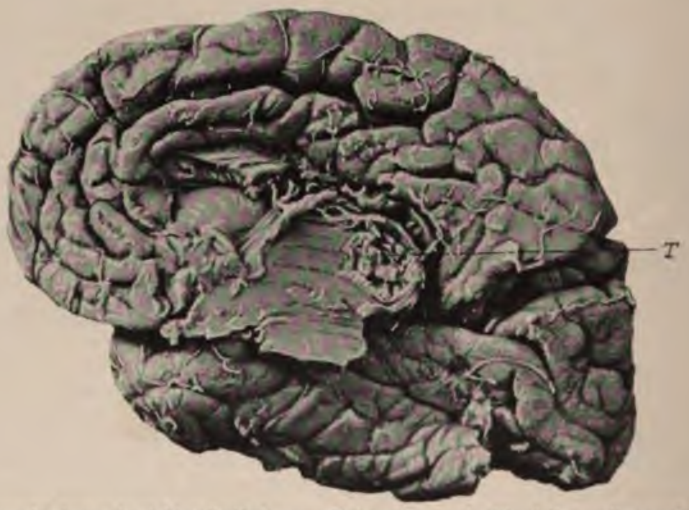
Author.	No. of cases.	Operable.	Reference.
Mills and Lloyd . . .	100	10	Pepper's System, 1886, vol. v.
Hale White	100	10	Guy's Hospital Reports, 1888.
Starr	300	16	Medical News, January 12, 1889 (children).
Knapp	40	2	Intracranial Growths, 1891.
Gray	53	4	Sajous' Annual, 1891.
Gray	49	6	" " 1892.
Seydel	100	3	Verhand. Deut. Gesell. f. Chir., 1892.
Dana	29	5	Trans. New York Acad. Med., Jan., 1893.
Starr	300	21	Brain Surgery, 1893 (adults).
Byrom Bramwell . . .	50	3	Edinburgh Med. Journ., June, 1894.
Oppenheim	23	1	Die Geschwultze des Gehirns, 1896.
Bruns	33	3	Encyclo. Jahresbericht., 1896.
Starr	100	20	Personal cases to 1903.
	1277	104	

Etiology. Little is known regarding the cause of brain tumor, though in a certain small percentage of cases a history of a blow or fall on the head has been obtained. It is known that males are more subject to brain tumor than females, and that persons of all ages are liable to brain tumor.

Pathology. **The Varieties of Brain Tumor.** Almost every variety of tumor known has been found within the brain. There are certain tumors, however, which are quite common, and others which are very rare. In the first class are tubercular, sarcomatous, gliomatous, gliosarcomatous, cystic, carcinomatous, and gummy tumors. In the second class are fibromatous, angiomatous, myxomatous, osteomatous, and lipomatous tumors, also psammoma, cholesteatoma, and echinococcus cysts.

Tubercular tumors of the brain are the most common of all the forms of tumor. They occur with greatest frequency in childhood, being sometimes primary, but usually secondary to tuberculosis of other organs. In 20 per cent of the cases recorded, tubercular tumors are multiple. This fact should not be forgotten in studying the symptoms of brain tumor and in diagnosticating the location of the tumor, for if the tumor is probably tubercular, and if the symptoms are not clearly explicable upon the theory of its location in one part of the brain, it is to be remembered that two or more different tumors in different locations may give rise to a great variety of symptoms. Tubercular tumors vary in size from a small collection of miliary tubercles lying in a mass of thickened pia mater up to a large solid circumscribed mass, with hard, cheesy, or broken-down granular centre and a distinct capsule. Not infrequently the tumor is surrounded by a tubercular infiltration both in the brain and its membranes. Sometimes irregularly-shaped deposits of tubercular tissues are found upon the base of the brain, in the meshes of the pia mater, compressing the cranial nerves. (Fig. 229.)

FIG. 229.

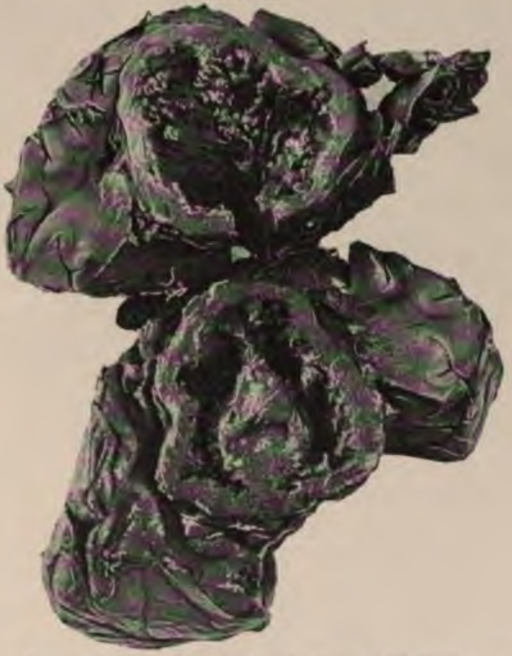


Tubercular tumor in right optic thalamus Tubercular meningitis of the median surface of the hemisphere.

Tubercular tumors may be found within the cerebral tissue at some distance from the surface, and no part of the brain can be said to be free from a liability to tubercular deposits. But the large majority of this variety of tumors are found in connection with the meninges and about the large vessels of the brain, so that they are more commonly discovered upon the surface and about the base than lying deep within the organ. When the tumor arises in the meninges meningeal thickening by tubercular deposits is quite common. In a few cases the cranial bones have been eroded in the growth of the tumor. Tubercular tumors are sometimes found in the brain after death when no cerebral symptoms have been present during life.[1]

The important facts to be considered in making the diagnosis of tubercular tumors are (*a*) hereditary tendencies of the individual to tuberculosis; (*b*) exposure to such influences as are known to favor the development of the disease; (*c*) the history of symptoms of primary pulmonary, or intestinal, or joint, or glandular affections, and (*d*) the presence of local signs or general evidences of tubercular infection.

FIG. 230.



Section through cerebrum, showing a large tubercular tumor with thick capsule and granular contents lying beneath the cortex in the centrum ovale of the frontal lobe.

When cerebral symptoms develop in an individual with tubercular disease the possibility both of tubercular tumor and of tubercular meningitis should not be overlooked.

Sarcomatous tumors are next in frequency to tubercular. They are rarely secondary to sarcoma in other parts of the body, and they are only multiple when they appear in the form of melanotic nodules, which are rare in the brain.

Sarcoma is usually encapsulated, round, or oval in shape, hard, approaching fibroma in consistency, and is easily separable from the brain tissue, which it compresses and indents more frequently than it infiltrates. Fig. 231 shows a sarcoma lying on one hemisphere of the cerebellum and Fig. 232 shows a sarcoma lying upon the base between the cerebellum and the pons. The relation of this tumor to the brain and the skull is shown in Fig. 233, which demonstrates the difficulty of reaching tumors of the cerebellum at an operation.[1] Round-cell and spindle-cell sarcomata are more frequently met with than myxosarcomata or gliosarcomata. These tumors grow rapidly and produce well-marked symptoms which do not vary very greatly in their intensity. They are not vascular, and hence the occurrence of apoplectic attacks during their growth is

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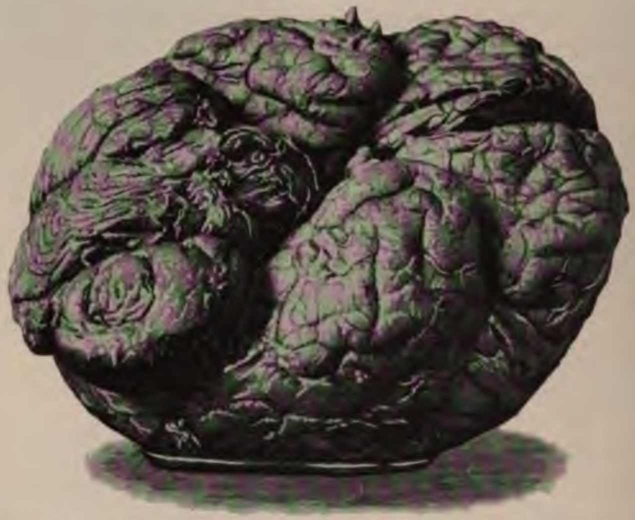
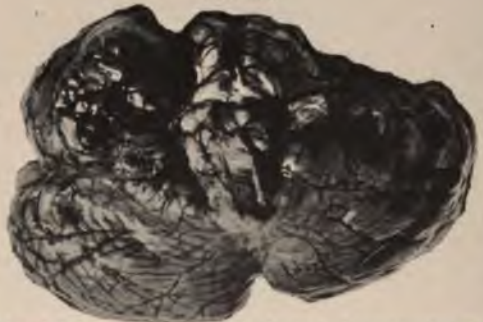
[1] Edinburgh Hospital Reports, vol. i. p. 420.

FIG. 231.



Sarcoma, encapsulated, with pedicle attached to the dura, lying in the posterior cranial fossa, and compressing the left hemisphere of the cerebellum.

FIG. 232.



Sarcoma, encapsulated, lying on the base of the brain and compressing the right side of the pons and right hemisphere of the cerebellum.

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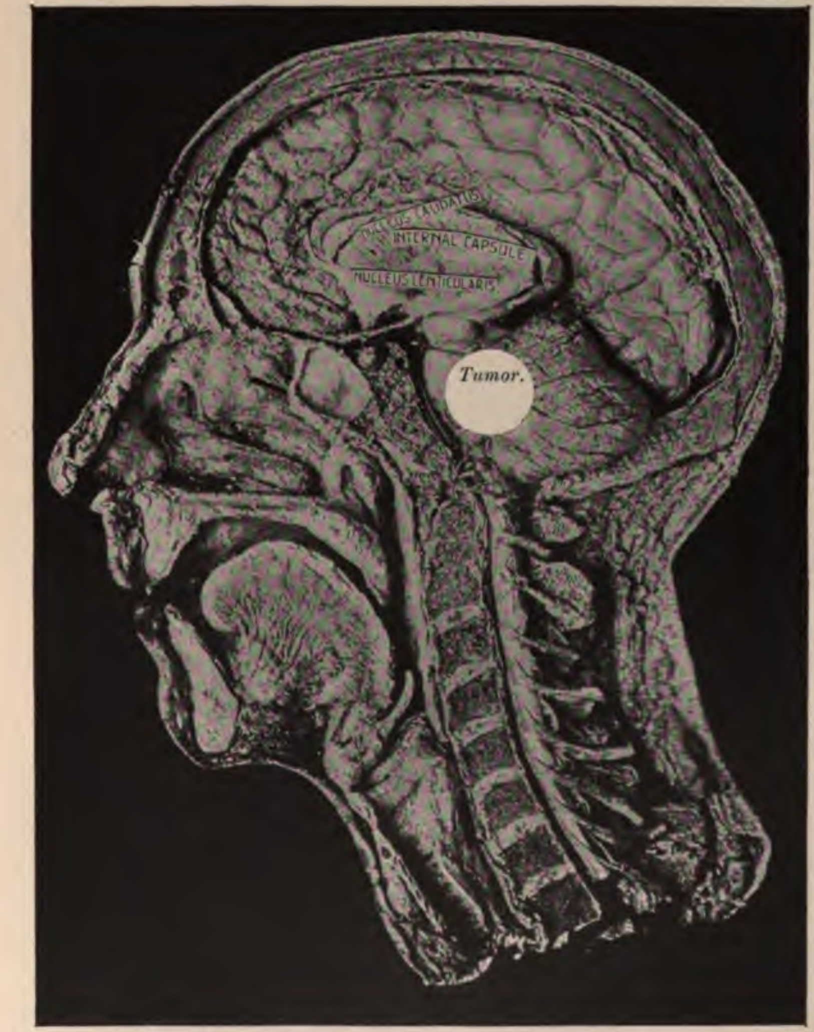
[1] American Journal of the Medical Sciences, April, 1893.

infrequent. It is not to be forgotten that a sarcomatous deposit in the meninges may occur, producing a thick layer of new tissue over an extensive surface of the cortex or base, and in such a case operative treatment is necessarily unsuccessful.[1] Occasionally a sarcoma has no defined limits, and infiltrates the tissue of the brain. Hence, sarcoma is not uniformly favorable for operation.

FIG. 233.



The situation of a tumor at the base of the cerebellum, shown on a Fraser photograph. The difficulty of removal is evident from its deep situation.

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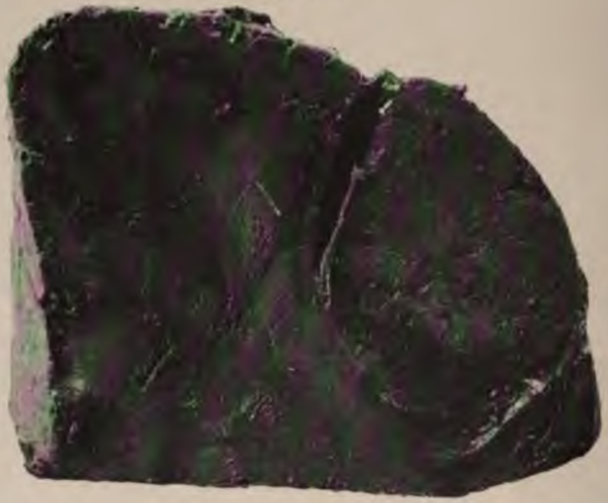
[1] Eskridge. Journal of the American Medical Association, September 30, 1893.

From the study of Table I. it is evident that sarcoma may be found in any part of the brain, but it is more frequent in the cortex and in the cerebellum than in any other localities. It is the form of tumor most easily extracted from the skull, and the majority of the successful cases of removal of brain tumor have been sarcomata.

Gliomatous tumors come third in the list. This tumor is a product of the neuroglia and presents the appearance of a connective tissue fibrillary network containing a greater or less number of large branching cells and many small nucleated cells.

Glioma of the brain is usually primary, but occasionally may develop secondarily to glioma of the retina.

FIG. 234.

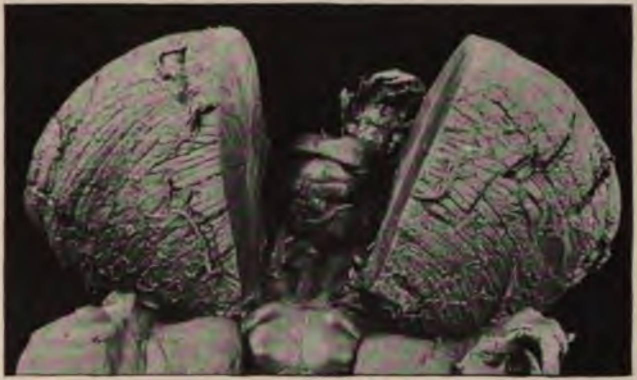


Glioma of the motor area infiltrating the cortex and centrum ovale, without distinct capsule, excepting at one part.

Gliomata vary in density, but are usually softer than sarcomata, and are rarely well defined or separable from the brain tissue, even those which appear to have a definite limit being found, on microscopic study, to be surrounded by a zone of gliomatous infiltration in the brain. The usual consistency of a glioma is about that of the brain substance. There is a tendency in all gliomatous tissues to undergo fatty degeneration, to break down and liquefy, so that cavities filled with clear fluid of a straw color or more sharply-defined cysts are very frequently found in and about a gliomatous tumor, and, in fact, some authors have maintained that all cysts found in the brain, not the relics of previous hemorrhages or of embolic softening, are due to gliomatous formations. A similar tendency to the formation of cavities within gliomatous tissue is manifested in the spinal cord in the disease syringomyelia.

Glioma may appear in any part of the brain, but is somewhat more frequent in the white substance of the brain than in the gray matter. As a glioma grows it destroys the brain tissue, its branching cells surrounding and annihilating both nerve cells and nerve fibres. It does not compress the brain as a sarcoma does.

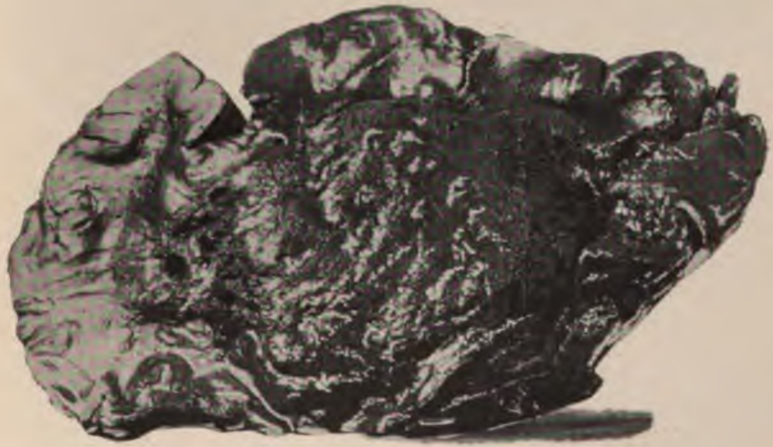
FIG. 235.



A glioma of the pons and medulla infiltrating the tissues, but not destroying the tracts. The symptoms were chiefly cerebellar.

Glioma is very vascular, and hence the symptoms which it produces are somewhat variable in intensity, and, in case the delicate vessels within it rupture, apoplectiform attacks occur.

FIG. 236.



Gliosarcoma of frontal lobes and corpus callosum. (Francis.)

Gliosarcoma is a variety of tumor not very frequent, partaking of the nature both of glioma and of sarcoma, commonly accompanied by cystic cavities. Like glioma the tumor cells are infiltrated through

The Gardner Sanitariu

the brain tissues; but like sarcoma, its constituents are spindle and round cells, and its density is considerable. It is rarely encapsulated, and, like glioma, is much more difficult of removal than sarcoma.

It is questionable whether the origin of a gliosarcoma is the development of sarcomatous cells infiltrating the brain, and causing an irritation and proliferation of the neuroglia, or whether there is a development of hard, round connective-tissue elements, sarcomatous in character, in the midst of the original glioma.

The symptoms are more closely like those of glioma than of sarcoma, the same variability of intensity being observed.

Carcinoma of the brain is usually secondary to carcinoma in other parts of the body, either of the orbit or retina, or of the internal organs or of the breast.

Carcinoma is a variety seldom found in children, and usually develops in adults above the age of fifty years. In cases in which it has occurred in children it has been from direct extension of the disease from the orbit. It may occur as the primary development of carcinoma in the body, giving rise to secondary infection elsewhere, but this also is rare.

Carcinoma grows rapidly in the brain, is not sharply defined, rarely has a capsule, is decidedly vascular, and, of all brain tumors, the one least open to operative treatment.

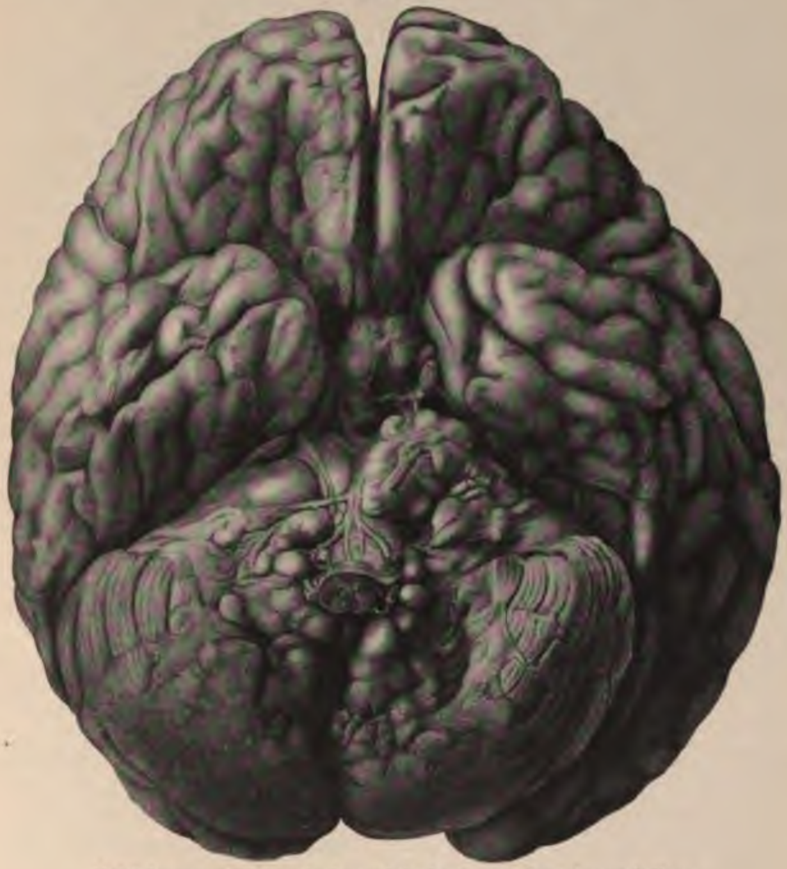
Cystic tumors of the brain may arise either in connection with glioma and gliosarcoma, as already described, or independently, as the result of parasitic infection. Hydatid cysts, echinococcus, and cysticercus are much more frequently recorded in German and Australian medical journals than in this country.

Kuchenmeister has made a collection of 88 cases of this variety, in all but 13 of which multiple cysts were found. Of these 49 involved the membranes, 41 were in the cortex, 19 in the white substance, 19 in the cerebellum, 18 in the ventricles, 17 in the basal ganglia, and a few in the corpora quadrigemina, pons, and medulla. These are not included in the tables, as it would give an appearance of undue frequency of this form of tumor. These parasitic cysts grow slowly and produce compression of the brain tissue, but do not destroy it. The symptoms which they produce are very rarely so well marked as to enable a diagnosis to be reached, but in cases in which a diagnosis is possible these tumors may be easily removed, as the cyst wall is separable from the brain tissue. Several such cysts have been removed in Germany.

Gummata of the brain, though very rare in childhood, and never the result of inherited syphilis, are very frequent in adult life, and may develop within a year of the original infection. They may also appear as the only apparent evidence of tertiary syphilis, even twenty years after the disease was acquired.

Gumma may occur as a soft gelatinous exudation upon the base of the brain, its favorite position, or anywhere in the membranes, or as a harder and more circumscribed tumor in the meninges, especially of the cortex and cerebellum. It grows rapidly, but also subsides rapidly under specific treatment. In one case under my care, where many symptoms, including double optic neuritis, were present, the patient was perfectly well in nine months. The occurrence of nocturnal headache and insomnia of an obstinate character in any case presenting the symptoms of brain tumor will suggest the probability of a gumma, and, in fact, in every case in which the diagnosis of brain tumor is made it is well to subject the patient to a course of treatment by mercury and iodide for a period of at least two months, in order to eliminate the possibility of gumma. Yet it must not be hastily argued that the subsidence of symptoms under this treatment is positive evidence that the tumor is syphilitic. In a case under my observation, which autopsy proved to be a small sarcoma with a large cyst, the wall of which was sarcomatous tissue, all symptoms, including optic neuritis, subsided under this treatment,

FIG. 237.



Multiple gummata upon the base of the brain and about the cerebellum.

and the patient remained well for a period of five months, and then sudden death in coma occurred unexpectedly.

Other varieties of brain tumor, such as fibroma, angioma, myxoma, psammoma, osteoma, cholesteatoma, lipoma, teratoma, which occur in the brain, are of great rarity. They occur both in children and in adults, and their diagnosis cannot be made from other forms of tumor during life.

Psammoma is usually the form of tumor occurring in the situation of the pineal gland, and gives rise to symptoms similar to those produced by tumors of the corpora quadrigemina.

Tumors of the pituitary body are not uncommon, and are usually of the nature of fibroma or myxoma. It is maintained by some that the disease acromegalia is the result of such tumors. It is not true, however, that a tumor of the pituitary body always produces symptoms of acromegalia, and in one case of acromegalia under my observation the autopsy failed to show any change in the pituitary body.

The appearance of a brain in which a tumor has developed is usually characteristic. The membranes are tense, the convolutions are flattened by pressure, the ventricles are distended by serous fluid, the brain tissue is wet, the weight of the brain is increased, and frequently about the tumor there is a zone of softening of greater or less extent, according to the variety of the tumor. Encapsulated tumors are less likely to be surrounded by a softened tissue.

Intracranial aneurisms present the same features as tumor of the brain. They are rarely large in size, they appear upon the larger arteries at the base of the brain and upon the Sylvian arteries. They are usually fusiform, occasionally round. They increase in size more rapidly than ordinary brain tumors, or than aneurisms elsewhere, and usually rupture before attaining any great size. They produce symptoms by their pressure and are occasionally to be diagnosticated by pulsating headache or the sensation which they produce, or by a distinct murmur audible to the stethoscope on the head. Such a murmur is not, however, pathognomonic, as I have heard such a loud double murmur over the Sylvian region in a case which proved at the operation to be one of extensive softening—no aneurism being found.[1] In a case recently seen at St. Luke's Hospital the aneurism developed rapidly after a blow on the head, producing, first, third nerve paralysis, and one month later right hemiplegia. The patient died three months after the injury, having had all the general symptoms of brain tumor. An aneurism of the left internal carotid artery at the point of union with the posterior communicating artery was found, which had ruptured. As the arteries were not diseased, it seemed probable that the bruise of the artery against the bone at the time of the injury had weakened the wall and predisposed to aneurism.

Symptoms. While the symptoms of brain tumor are in many cases very clear and characteristic, so that there need be little doubt in regard to the diagnosis, both of the existence of a tumor, of its nature, and of its location, it must be stated that there are other cases in which no characteristic symptoms appear at all.

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[1] Starr. Brain Surgery, Case xix.

Numerous cases have been recorded in which large tumors have been found unexpectedly after death, even in locations in the brain in which the existence of a tumor would presumably have produced very marked symptoms. Thus, in one case under my observation, a large glioma occupied the entire white substance of the frontal lobe upon the left side, yet the patient had suffered only from occasional attacks of epileptic convulsions, not preceded by an aura, and never unilateral, was as intelligent as ever up to the time of his death, though his family had noticed some irritability of temper at times, and on this account had occasionally questioned his responsibility for certain peculiarities. This man was under observation by most competent physicians for several years, and was supposed to have epilepsy. His eyes were examined and found to be normal within a few weeks of his sudden death, which took place in a convulsion. He had never had the ordinary symptoms of a brain tumor. Schoenthal[1] has recorded a case of supposed hysteria in which the variability of the symptoms was surprising, and in which careful examination failed to reveal any permanent or tangible physical signs. After death a large tumor of the right frontal lobe was found. Mayer[2] has collected several such cases. Buzzard has recorded similar cases. Bramwell[3] has described an enormous tumor lying in the central region upon the cortex of the right side in which there were no symptoms of paralysis whatever. And many tumors have been found in and about the cerebellum which have failed to produce the ordinary symptoms of brain tumor.

Tumors upon the base of the brain may also be found involving structures which, during life, appear to have been capable of carrying on their ordinary functions. Thus a child under my observation suffered for five months from occasional convulsions, from headache, from spastic paraplegia, but had no difficulty whatever of vision, and no paralysis of the cranial nerves. The autopsy showed a large tumor upon the base in the median line, through which both the optic nerves passed and in which the optic chiasm lay. It must have rested upon all the motor nerves of the eyes and compressed the fifth nerve on both sides. This tumor had grown upward, filling both the lateral ventricles and obliterating the third ventricle entirely.[4] (Fig. 238.)

I have seen a tumor of the medulla oblongata, an infiltrating glioma, which produced an apparent uniform increase in size of the entire medulla to double its ordinary dimensions, but in which there were absolutely no signs of any disease of either cranial nerves or tracts passing through this important part of the nervous system. The patient had headaches, slight optic neuritis, occasional convulsions, and a slightly staggering gait, which led to the diagnosis of tumor of the cerebellum. (See Fig. 235.)

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[1] Berliner klin. Woch., 1891, No. 10. See also Williams, Lancet, October 14, 1893.

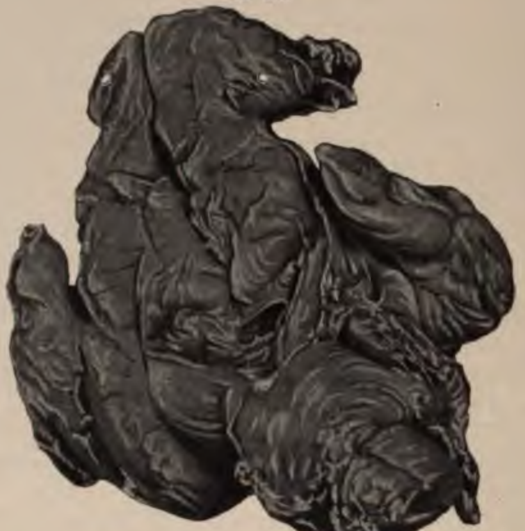
[2] E. Mayer. Inaug. Dissert., Berlin, 1891.

[3] Intracranial Tumors. p. 12.

[4] Transactions Amer. Neurol. Assoc., 1891. Starr, Brain Tumor.

It is thus evident that tumors in the various parts of the brain substance may develop and assume a considerable size without producing characteristic symptoms either of a local or of a general type. Such cases, however, are rare, and the careful observer will usually be able to diagnosticate a brain tumor.

FIG. 238.



Sarcoma of base separating the crura and surrounding the optic chiasm and greatly stretching the optic nerves. The exit of the optic nerves from the tumor is indicated by the white dots.

The symptoms of brain tumor are very numerous, but for purposes of convenience may be separated into two categories.

I. General symptoms due to the existence of a new-growth irrespective of its position. These are headache, general convulsions, double optic neuritis, and optic atrophy, changes of disposition and of mental power, vomiting, vertigo, insomnia, changes in the pulse rate, attacks of syncope, polyuria, and progressive malnutrition.

II. Focal symptoms of the disease in the cortex of the brain or beneath the cortex in the projection tracts which join the cortex to the various subcortical centres. These symptoms are unilateral spasms, monoplegia, or hemiplegia, paræsthesia or anæsthesia in one or more limbs, hemianopsia, and the various forms of aphasia. Affections of the cranial nerves and basal parts of the brain, which occur with tumors in the basal ganglia and cerebral axis, or external to the brain upon the base. These latter symptoms are frequently very complex, as may be supposed, when it is remembered that the twelve cranial nerves have extensive nuclei of origin and a long course, and that all connection between the external world and the brain passes through the cerebral axis.

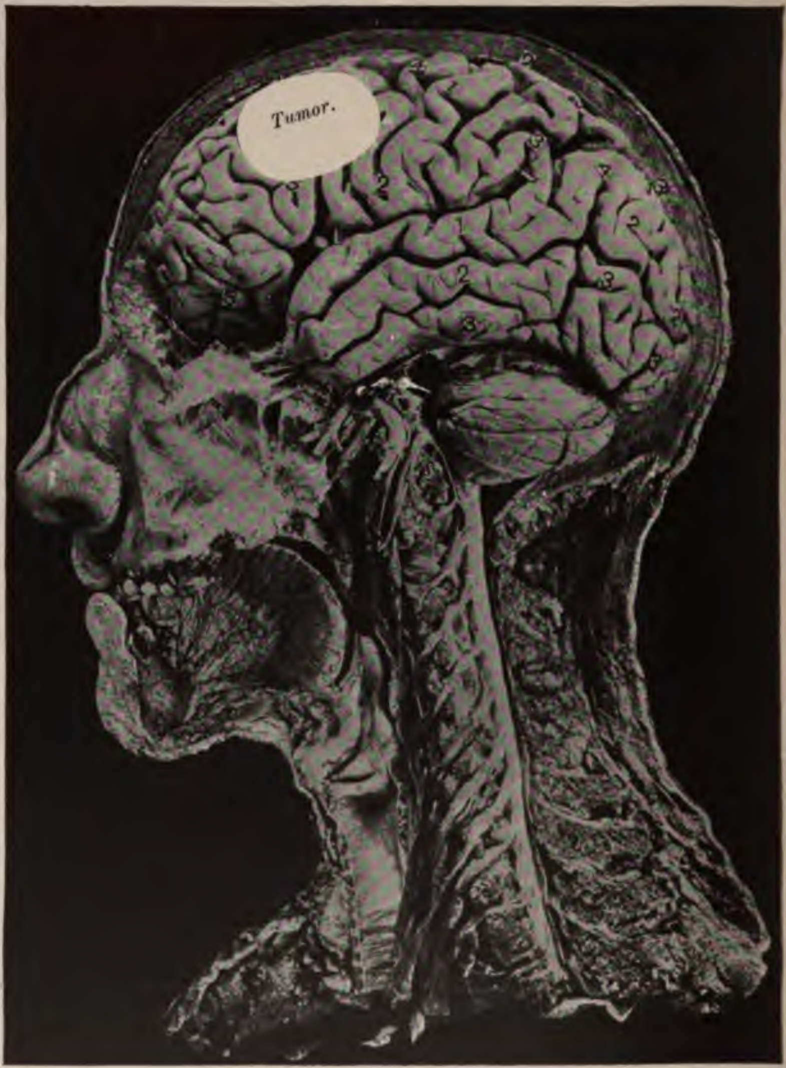
In the vast majority of cases of brain tumors we find some of these general and focal symptoms present. And from their combination and the order of their development it is usually possible to arrive at a definite diagnosis. These symptoms must, therefore, be studied with care.

I. General symptoms occur irrespective of the location of the tumor and depend upon its rapidity of growth, its vascularity, and its pathological character. They vary in severity from time to time probably in accordance with the activity of the pathological process going on in and about the tumor and with the condition of blood supply in the brain. When a tumor is growing rapidly they are very severe; if it remain stationary they may almost disappear. They are often affected by those agents which produce a temporary cerebral hyperæmia or anæmia, as is proved by the fact that hot foot baths or general hot baths may produce marked changes in the degree of general symptoms.

Headache is the most prominent and constant symptom of brain tumor. It varies in severity, but is usually very intense. It may be intermittent, it may recur with periodicity, is usually worse at night in syphilitic cases, worse on waking in other cases, is increased by physical effort or mental strain, or emotional excitement. It may be a dull, heavy, continuous pain, with sharp paroxysms, during which the patient cannot control his suffering. It varies in its location, is usually frontal or occipital, and the situation of the pain rarely indicates the situation of the tumor. If, however, the pain is constantly located in the occipital region the tumor is probably in the posterior fossa. The pain is often associated with indefinite cerebral sensations described as fulness, pressure, confusion, tightness, as if a band were drawn about the forehead, and these give rise to great discomfort. In infants the existence of headache may be inferred from constant motion of the head, from movements of the hands grasping the head, or pulling the hair, and from sudden outbursts of screaming without other ascertainable cause. Headache is probably less severely felt in cases of tumor in infants, as the opening of the sutures may prevent the extreme degree of intracranial pressure to which the headache is chiefly ascribed. In adults the headache prevents the patient from making any physical or mental exertion, and leads to a desire for seclusion and quiet.

The causes which are supposed to produce the headache are the increase of the intracranial pressure and the consequent stretching of the membranes; the existence of effusion into the ventricles, which is a frequent accompaniment of a tumor; the variations in the cerebral circulation produced by the pressure of the tumor, or the direct involvement of the sensitive dura mater and branches of the fifth nerve. Such effects of the presence of a tumor are more likely to follow when the new-growth is in the posterior cranial fossa under the tentorium cerebelli, and in such cases headache is most constant and most severe.

FIG. 239.



Situation of a tumor, shown in a photograph from Fraser's *Guide to Cerebral Operations.* The size is in exact ratio. The tumor was removed by McBurney. This figure demonstrates the relation of the convolutions of the cerebrum and the cerebellum to the head and the exits of the cranial nerves.

The headache is often associated with a marked tenderness of the head to percussion, and if this is not due to sensitiveness of a single nerve trunk it is a very valuable sign of the location of a tumor.

Such a tenderness to percussion is more commonly found in tumors lying just beneath the bone, that is cortical tumors, than in tumors deep within the brain. It has been asserted that a flatness to auscultatory percussion can be detected over a tumor, and I am able to confirm this statement, having noticed it in several cases. It is not always present.

General convulsions are the next most frequent symptom of brain tumor. They are particularly liable to occur as an early symptom in children, but may also be the first symptom of tumor in adults. Thus, in a patient of mine from whom McBurney removed a large sarcoma of the frontal lobe, the first symptom was a general convulsion which occurred two months before the headache began, and was never repeated during the three years' course of the disease.[1] (See Fig. 239.) In another patient under my care, who subsequently developed all the general symptoms of brain tumor, general convulsions occurred at intervals during three months before any other symptom appeared. A case diagnosticated as epilepsy has already been mentioned.

It is not uncommon to find convulsions occurring at irregular intervals from the onset of the disease to its termination. They usually occur at long intervals, but have been known to be as frequent as twenty to thirty a day. They may be slight in degree, almost of the nature of petit mal, with a sudden loss of consciousness, or this loss of consciousness may be attended by a little twitching of the face and eyes, with stiffening of the back and extremities, balancing movements of an automatic kind, which prevent falling, and then recovery, or they may have all the general characteristics of a general epileptic seizure followed by coma. Sometimes a peculiar general tremor follows the attack, and may last for an hour or more.

General convulsions may be a culmination of a local spasm; hence, wherever a convulsion occurs particular attention is to be called to the manner of its beginning, and the patient is to be carefully observed and instructed to watch for a conscious aura. The significance of local spasms will be discussed in the next section.

Convulsions are usually indicative of rapid progress in the new-growth, of effusion into the ventricles, of hemorrhage within the tumor or of a secondary affection of the meninges. They may occur from tumors situated anywhere, and have no significance as a diagnostic symptom of the location. Death not infrequently occurs in convulsions in brain tumor, and hence a development or rapid increase of this symptom is a sign of danger.

Changes of disposition and of mental power occur in the course of brain tumor with considerable frequency.[2] These have sometimes been explained by the existence of headache, the suffering producing mental exhaustion. But they are also observed quite uniformly with tumors of the brain, even when headache is not severe. In children this symptom is quite noticeable, the child becomes fretful and irritable, refuses to notice its toys or to play, or, if it does so, soon becomes wearied, and requires constant attention. It may become indifferent to things in which it was formerly interested, prefer to lie down and to keep quiet, in a manner unnatural to a healthy child, and may even become somnolent and lethargic, or it may have apparent attacks of causeless terror. In adults the mental dulness is very noticeable. The patient takes little interest in his ordinary surroundings or business, and is content to sit or lie for hours doing nothing and apparently with a vacant mind. Such a patient is easily aroused and replies intelligently to questions, but cannot be considered in a normal state of mental activity. On the other hand, such a patient is not demented, although late in the course of the disease a condition approaching dementia is quite often observed, especially in tumors of the frontal lobes. Attacks of maniacal excitement have been recorded, but are very rare. The usual mental state present in brain tumor, after it has existed for a period of six months may be described as one of apathy. These symptoms are probably referable to the increased intracranial pressure and consequent compression of the brain, which interferes with the processes of association upon which all thought depends. The tumor appears by its pressure to hamper the transmission of impulses throughout the brain. Somnolence may be associated with this apathy. In the case of a tumor of the frontal lobes and corpus callosum[1] mental apathy was the chief symptom from beginning to end. (See Fig. 236.)

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[1] See American Journal of the Medical Sciences, January, 1893.

[2] Schuster. Psychische Störungen bei Hirntumoren, Stuttgart, 1902.

Double optic neuritis and optic nerve atrophy are very important diagnostic symptoms of intracranial tumor. Neuritis is usually associated with other signs of increased intracranial pressure, but may occur without such pressure. It is present in 80 per cent. of the cases and should be looked for in every case which presents cerebral symptoms. A marked degree of optic neuritis may exist without any impairment of vision; hence, the ophthalmoscope should be used, whether the defect of sight be present or not. But when the patient shows impairment of visual power or limitation of the visual field for colors or for light, or is becoming blind, it will be found that optic neuritis or optic atrophy is fully developed. Sudden loss of vision appears to be more commonly noted in histories of children's cases than in those of adults, possibly because a gradual loss of sight is not detected. It is true that hydrocephalus may cause choked disks, and hence from this symptom alone a tumor cannot be diagnosticated. But in cases where the diagnosis is difficult no more important objective evidence of brain tumor can be found.

Tumors of the cerebellum and corpora quadrigemina and tumors upon the base of the brain and in the basal ganglia produce optic neuritis more constantly and earlier in their course than tumors situated in the cortex or centrum ovale. Optic neuritis is usually double, though it always appears first in one eye, and is rarely equally intense in both eyes; but in a few cases it has been found in one eye only, and then is thought to indicate disease of the nerve in the orbit or in front of the optic chiasm, rather than a distant tumor. For the exact changes in the retina and for the pathological causes of optic neuritis the reader is referred to Chapter XXXIV.[1]

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[1] R. G. Francis. American Journal of the Medical Sciences, June, 1895.

Vomiting is a symptom of brain tumor more frequently observed in children than in adults. It may or may not be accompanied by nausea. It may occur accidentally, without special relation to the time of meals, or it may be so continuous as to threaten inanition. It occurs not infrequently on any movement of the head after the patient has been confined to bed for some time, and then it is usually associated with vertigo. It also frequently accompanies severe headache.

Vertigo is sometimes a coincident symptom, but usually occurs independently of vomiting. The patient feels dizzy, feels himself turning or falling, and things about him appear to be in motion. He grasps at near objects for support, covers his eyes with his hands or lies down on the floor and cries out with bewilderment and distress. Like vomiting, vertigo may be excited by changes of position. It occurs at intervals in attacks of short or long duration. It occurs more frequently with tumors in the posterior fossa, in the cerebellum, or pons, or on the base involving the auditory nerve, than with tumors elsewhere. Such attacks of vertigo are not to be confounded with the slight constant vertigo due to double vision, and secondary to paresis of the third and sixth nerves.

Insomnia may be due to disturbances of the cerebral circulation or to the intensity of the other general symptoms, and is much more rarely complained of in cases of tumor in children and youth than in adults suffering from syphilitic tumors.

Fever and changes in the rapidity and rhythm of the pulse have been observed in the course of brain tumors. The former is ascribed to inflammatory changes in the brain or meninges as a complication. The latter is regarded as evidence of increased intracranial pressure. Slow and irregular pulse is the form usually noted, but toward the close of life very rapid pulse has been observed. Irregular or Cheyne-Stokes respiration has also been noticed as a terminal symptom.

Occasionally attacks of syncope occur in patients with tumor of the posterior fossa, and a general feeling of weakness is not infrequently complained of. Polyuria and glycosuria may develop in the course of brain tumor[2] as a symptom of increased pressure. It has been found also in small tumors of the medulla[3] as an evidence of irritation of the vagus nucleus.

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[1] See especially Knies. The Eye in General Diseases. William Wood & Co., 1895.

[2] Rothmann. Zeitschrift f. klin. Med., 1893, xxiii. p. 339.

[3] De Jonge. Arch. f. Psych., xiii. p. 658.

A rise of temperature in the scalp over the tumor has been observed in a few cases, but is not a uniform or reliable symptom of brain tumor.

The combination of several of these general symptoms in any case should lead to a strong suspicion of intracranial tumor. When, in addition, local symptoms are added, the diagnosis should not be difficult. In all cases the onset of the general symptoms is gradual, their intensity increasing as time goes on, and a careful study of their mode of development and progress is a great aid in the diagnosis of the kind of tumor present, of its size, and of the rapidity of its growth.

The distress produced by the general symptoms is usually much greater than that caused by the local symptoms, and consequently treatment is often required for their alleviation.

The focal symptoms of brain tumor depend entirely upon the situation of the tumor.

A distinction is made between direct and indirect focal symptoms, the first being due to irritation or destruction of a limited area by the tumor, the second being due to interference with the function of an area by disease at a distance from it which impairs its circulation or causes pressure upon it. Thus, a tumor of the cerebellum may cause inco-ordination and vertigo as direct symptoms, and also cause paralysis of the sixth and seventh cranial nerves as an indirect symptom of displacement of the pons Varolii to one side, which stretches these nerves unduly. Tumors usually cause both forms of focal symptoms, and, therefore, much care must be given to the question of their significance in any case.

Focal symptoms may further be divided broadly into two classes, those produced by tumors in the cortex and cerebral hemispheres and those produced by tumors upon the base of the brain affecting the cranial nerves. Reference may be made to the preceding chapter upon the diagnosis of brain diseases for a special consideration of these various symptoms. With the anatomy of the brain in mind it is not difficult to understand the various local symptoms that may arise from the existence of brain tumor in different localities. It should be remembered, however, that a tumor of very slow growth, which compresses and does not infiltrate the brain may exist in any location without necessarily producing focal symptoms, until it has attained a considerable size. It is evident that a single tumor rarely, if ever, produces a large number of focal symptoms but that all tumors lying in any given location will produce the same symptoms. It is also evident that tumors will almost always produce unilateral symptoms, inasmuch as they rarely lie exactly in the median line. And even the tumors of the corpus callosum, of which about twenty have been recorded, have rarely been so exactly median in their situation as to give rise to symmetrical symptoms upon both sides.

The focal symptoms usually commence gradually and are limited in extent, but increase steadily as the tumor grows.

The focal symptoms of tumors of the frontal cortex may be enumerated as follows: mental inactivity, forgetfulness, lack of judgment, decided change in character, irritability of temper and unusual stupidity, an inability to concentrate the attention, to think connectedly and continuously, to learn easily, to exercise self-control, lastly a state approaching mild dementia without delusions, in which the patient may become dirty and disregard all restraints of decency. These symptoms are especially marked in tumors of the frontal cortex and subjacent white matter.[1] They are not present in tumors lying upon the base of the frontal region on the orbital bone. They rarely appear in tumors in other regions of the cortex until the last stage of the disease, when the intracranial pressure is very great. A decided change in character and disposition, a mental apathy, and a tendency to somnolence must be regarded as a local sign of frontal lobe disease. While the integrity of the frontal lobes is necessary to mental action, yet no special loss of any mental faculty can be said to result from their destruction. Nor is there any apparent way to detect from the mental symptoms in which hemisphere of the brain the tumor lies. It is true that much of our thought is dependent upon the integrity of our speech centres, and hence the lesions of the left hemisphere, if they interfere with the use of language, or even of the recollection of words, appear to give rise to mental confusion, which is not so noticeable in lesions of the right hemisphere. But it is not possible to locate all mental action, as has been done by Phelps,[2] in the left frontal lobe. A consideration of the facts known regarding the wide dissemination of memory pictures over the entire hemisphere, and their fundamental importance in all thinking is sufficient to prove that mental activity is not a function of any one region of the brain. (See page 462.) The diagnosis of tumor of the frontal lobes is, therefore, rarely made from a study of direct focal symptoms. It is to be remembered, however, that a tumor when situated in other regions of the cerebral hemispheres, excepting only the temporo-sphenoidal lobes, will produce other direct focal symptoms, the absence of which may lead to the suspicion that a tumor is situated in the frontal lobe.

Tumors in the frontal lobe frequently produce irritation of the cortex. While such irritation is limited to the frontal cortex we have no evidences of its occurrence, but there is a tendency for irritation starting in one part of the brain to radiate outward to adjacent parts, like the ripple upon a lake when a stone is thrown in. When irritation starts from the frontal region it frequently appears to radiate backward, and thus involve the central region of the brain, and under these circumstances symptoms referable to the central region appear, namely, localized spasms, paræsthesia, and paralysis.[3]

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[1] Ferrier. Lancet, June 4, 1892.

[2] American Journal of the Medical Sciences, April, 1902.

[3] See a case of my own, shown in Fig. 239.

Bruns[1] has recently recorded three cases of frontal tumor in which there had been some disturbance in the balancing power and a staggering gait similar to that observed in cerebellar disease; but this symptom has not been uniformly noticed. It is known that the frontal lobes and cerebellar hemispheres have a crossed connection, and it is possible that this symptom may prove of some diagnostic value if confirmed. It may have been due to an irritation of the cerebellum conveyed along these connecting tracts.

Tumors situated in the third frontal convolution of the left hemisphere in right-handed persons and of the right hemisphere in left-handed persons produce motor aphasia with agraphia, the patient being able to understand language both written and spoken, but being unable to give expression to his ideas. When this symptom develops as the result of brain tumor the disturbance of speech is gradual in its onset, the patient noticing first a hesitancy in speech, a loss of words, and possibly a misuse of words before he loses the power of expression. The aphasia is less complete in brain tumor than in those apoplectic conditions which are the common cause of aphasia.

Tumors involving the motor area of the brain give rise to well-marked and distinctive symptoms. The special functions of the motor area are displayed in Figs. 160 and 161, and the symptoms that are produced by a tumor beginning in any portion of this area are easily deduced from reference to these figures. If the tumor is cortical in its situation it almost inevitably gives rise to local symptoms; these may be in the form of tonic or clonic convulsions, occurring at intervals, either limited to one part of the body, such as the face, or hand, or foot, or extending from the part first invaded to other parts in a definite order of succession, the extension being commonly from face to arm, to body, and then to leg, or in the reverse order. Such attacks, first described by Hughlings Jackson, are known as attacks of Jacksonian epilepsy. If it be remembered that the irritation starting from one part of the cortex and radiating outward to other parts produces a spasm beginning in the muscles represented in the centre first irritated, and extending to other muscles represented in adjacent centres, it will be evident that the exact order of the spasm aids one to determine the situation of the tumor, for it is evident that a spasm beginning, we will say, in the thumb and extending then to the fingers, to the hand and up the arm to the shoulder, will indicate an initial irritation of the thumb centre of the cortex, extending then to the centre for the fingers, hand, and arm. A convulsion beginning with the turning of the head and eyes to one side indicates a tumor situated in the anterior middle portion of the motor zone, and if that irritation extends from this point backward or downward the spasm will extend from head and eyes to arm or face, respectively. The situation of several tumors which produced such local spasms is shown in Figs. 162 to 170, pages 422 to 424.

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[1] Deutsche medicinische Wochenschrift, 1892, p. 138.

Therefore, the point of beginning of a spasm and its order of extension are the most important localizing symptoms of brain tumor in the motor area. Such a spasm is usually followed by temporary paralysis, and as the tumor increases in size and the spasms increase in frequency and extent, the paralysis may remain permanent between the spasms. Spasms due to cortical irritation are almost invariably attended by numbness and tingling in the part first affected by spasms, so that such a tingling is often a valuable indication of the location of irritation in the cortex. This tingling may even precede the spasm, and has been termed by Seguin the "signal symptom" of a cortical irritation. When such a paræsthesia is a permanent symptom the tumor is more likely to be situated behind the fissure of Rolando than in front of it. When the irritation produced by a tumor in the motor zone is very intense, the local spasm after extending from one limb to the entire side of the body may even become general, be associated with loss of consciousness, and terminate as a general convulsion. While such an order of extent preceding the general convulsion is of great diagnostic importance, a general convulsion beginning suddenly, with loss of consciousness and without any local spasm, is not to be regarded as indicative of disease in the motor area. There are cases of brain tumor in which the motor area has been generally compressed and destroyed without the production of these local spasms, but with a progressive and increasing paralysis alone. It is thought that such a paralysis not associated with spasms indicates that the situation of the tumor is in the white matter beneath the cortex and not in the cortex itself, and that the symptoms are due to an invasion of the motor tract in its passage toward the internal capsule. Some ataxia is commonly associated with the paralysis produced by tumors, and a marked inco-ordination or disturbance of the muscular sense, like anæsthesia, points to a situation of the tumor behind the fissure of Rolando. The condition of the deep reflexes is altered in all cases of tumor in the motor area; a marked increase in the tendon reflexes at the elbow, wrist, knee, and ankle is one of the early signs of paralysis. There is no atrophy in the paralyzed muscles, but merely a slight wasting from disuse, and there is no change in the electric reaction of the muscles. There should be no difficulty in differentiating a cerebral paralysis, even of the monoplegic type, from spinal or nerve trunk paralysis, even in infancy, because of these points of contrast. The careful study of the localizing symptoms of tumors of the motor region has resulted in successful localization of such tumors and in their successful removal from the brain; in fact, there is no region in the brain in which a tumor is more easily accessible or more possible of early diagnosis than in the motor area.

Tumors of the parietal region, including the superior and inferior parietal lobules are not uniformly attended by distinctive and local symptoms, but in a considerable number of cases disturbances of sensation and of muscular sense in the limbs of the opposite side have been observed. It has been stated already that tumors in the posterior central convolution are more liable to produce sensory symptoms than those in the anterior central convolution. And it is possible that tumors lying in the parietal region have caused sensory symptoms by pressure upon the sensory tracts on their way from the posterior portion of the internal capsule to the central region of the brain. If this is so, then ataxia and anæsthesia appearing in connection with a tumor of the parietal region would be an indirect local symptom rather than a result of a lesion in the sensory centres themselves. The question of the existence of a muscular sense as distinct from the sensory centres and the motor centres has been discussed on page 434.

The symptom of word-blindness is almost uniformly due to a lesion of the inferior parietal lobule in the left hemisphere in right-handed people and in the right hemisphere in left-handed people. A reference to Plate XIX. and to the discussion of aphasia on page 452 will show the importance of this symptom.

Tumors lying in the lower half of the parietal region are necessarily near to the visual tract which passes outward from the internal capsule to the occipital lobe, and hence, defects of vision of the form of bilateral homonymous hemianopsia have been recorded in connection with tumors in this locality.

Tumors of the occipital region of the brain give rise to the important local symptom of lateral homonymous hemianopsia. (See page 449.)

Henschen's researches show that a lesion at almost any portion of the occipital lobe, if sufficiently deep to compress the white matter of this region, will produce hemianopsia, and it is to be expected that in any lesion of the nature of a tumor in which small areas of tissue are not destroyed, but considerable areas are primarily affected and adjacent regions are strongly compressed, symptoms of hemianopsia will be frequent.

The occipital lobe is easily accessible to the surgeon, and therefore it is important that tumors here should be discovered as early as possible. It is not generally appreciated that hemianopsia is a symptom often entirely overlooked by a patient, a case having been recently reported by Bleuler[1] in which, though well-marked hemianopsia existed, the patient was entirely unaware of any visual defect. It is therefore exceedingly important that the extent of the visual field in both eyes should be carefully tested in every case of suspected brain disease, each eye being tested separately.

Lesions of the occipital lobe are capable of producing a disturbance known as blindness of mind, in which the patient, though seeing an object no longer, recognizes it as having been previously known. This is a condition allied to word-blindness, in which the patient is suddenly deprived of the power of reading. This condition appears to be more frequent in lesions upon the left side of the brain in right-handed persons, and upon the right side of the brain in left-handed persons. It may also occur when a lesion is entirely subcortical, as well as when it is in the cortex of the occipital lobe. This symptom has been discussed in connection with aphasia (page 450).

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[1] Archiv f. Psychiatrie, vol. xxv. p. 39.

Tumors of the temporal lobes or tumors lying upon the base of the cerebral hemispheres above the tentorium or upon the petrous portion of the temporal bone give rise to few recognizable symptoms. In a few such tumors disturbance of taste and smell seem to have been present when the apex of the temporal lobe had been invaded by the growth, and when the uncinate gyrus had been destroyed. There are no known symptoms produced by disturbance of the lingual lobule.

Tumors invading the first and second temporal convolution upon the left side in right-handed persons and upon the right side in left-handed persons produce the form of aphasia known as word-deafness, in which the person is unable to recall the names of places or persons, and cannot understand what is said to him. They may also cause intercortical-sensory-aphasia. (See page 466.)

It is believed that the first and second temporal convolutions are the termination of the auditory tract, and that each hemisphere is related to both ears. Deafness of cerebral origin is, however, rare, and is not often observed in tumors of these convolutions.

Tumors lying within the Sylvian fissure and upon the island of Reil produce numerous symptoms on account of their pressure upon adjacent parts; thus, by pressing upon the third frontal convolution they cause motor aphasia; by invading the operculum they produce facial paralysis; by pressing upon the inferior parietal lobule or the superior temporal convolution they may produce various symptoms of sensory aphasia, and by direct pressure upon the island of Reil and upon the underlying external capsule, they may cause paraphasia. If this pressure is transmitted more deeply to the internal capsule symptoms of tumor in this region may also be caused.

Tumors lying in the median surface of the brain, upon the gyrus fornicatus, gyrus marginalis, or upon the hypocampal convolution are not easily diagnosticated. Ferrier believes that in the latter convolution are situated the tactile centres. The majority of physiologists (Munk, Horsley, Schaeffer) and of pathologists deny this, and recently a case of glioma of this region without sensory symptoms has been reported by Humphrey.[1] Tumors in this region are of rare occurrence, and no focal symptoms can be assigned to them as yet.

Tumors of the corpus callosum are not at all common, only twenty cases being so far recorded in literature.[2]

The symptoms in a majority of these cases have been chiefly general symptoms, viz.: headache, vomiting, optic neuritis, epileptiform attacks, physical inertia, hemiparesis, and disturbance of intelligence, principally in the form of dementia. These were the symptoms in one case under my observation. The last-named symptom is the only one that appears to be of constant occurrence in these cases, and in many of them it has been preceded by symptoms quite comparable to those of hysteria. The majority of cases of tumor of the corpus callosum have therefore resembled tumor of the frontal lobes. When tumors occur in the corpus callosum they are more likely to be situated far forward about its knee, rather than in its thinner posterior part. (See Fig. 234.)

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[1] Brit. Med. Journ., Aug. 27, 1893.

[2] Bruns. Berliner klin. Woch., 1886, Nos. 21 and 22. Bristow, Dis. of the Nervous System, p. 271. Lutzenberger, Neurol. Centralb., 1890, p. 251. Berkley, American Journal of the Medical Sciences, June, 1890. Oliver, University Medical Magazine, April, 1891. Francis and Starr, American Journal of the Medical Sciences, June, 1895.

Our knowledge of the function of the corpus callosum is very imperfect, though it is evident from its structure that it associates the action of the two hemispheres. The corpus callosum has been wholly wanting in the brain in a few cases, and although this is occasionally associated with imbecility it is a condition which has been found in persons who presented no symptoms during life.[1] It is evident, therefore, that tumors in this locality cannot be positively diagnosticated, and the situation of such tumors deep between the hemispheres forbids the possibility of their removal.

Tumors of the basal ganglia, including the lenticular and caudate divisions of the corpus striatum and the optic thalamus, are not very rare. Our knowledge of the functions of these ganglia is very imperfect, and the necessary loss of function which must occur when they are destroyed by softening cannot be detected during life. It has been suggested that these ganglia are to be regarded as vestigial bodies, like the appendix vermiformis, without active function in man. Tumors, however, in this region, almost always produce pressure upon the internal capsule through which pass the most important motor and sensory tracts, and which lies between these ganglia. Hence symptoms of the nature of hemiplegia, hemiataxia, hemianæsthesia, and hemianopsia are observed when the tumor invades one or more of these tracts. (See Fig. 178.) It may then be possible to detect the progressive growth of a tumor here by the succession of symptoms produced, one tract after another being invaded by the disease. Motor symptoms are more common in cases of tumors of the corpora striata. Sensory symptoms occur in cases of tumors of the optic thalami. When a tumor invades the posterior part of one thalamus, homonymous hemianopsia is an invariable symptom. This will be easily understood by reference to Fig. 183.

It has been supposed that lesions in the optic thalamus give rise to disturbances in the recognition of the position of the limbs of the opposite side, and that in consequence awkward positions are assumed and awkward movements, athetoid in character, are made (Meynert). This I can confirm. In the patient whose tumor of the optic thalamus is shown in Fig. 240 there was noticed a peculiar forced posture of the limbs when he was at rest. The leg was thrown back and out with the toe pointed down, and the hand was held up above and in front of the head. This symptom persisted for many weeks until total hemiplegia gradually developed. It has also been affirmed that a tremor quite similar to that of multiple sclerosis may be caused by a tumor of the thalamus.[1] It has been noticed that disturbances in facial expression and in the natural play of facial feature in speech and thought have been noticed in patients suffering from disease of the optic thalamus.[2] The face is affected on the side opposite to the lesion and does not act in smiling, but can be moved voluntarily. It has been thought that certain inhibitory functions are exercised by the thalamus, and that undue emotional reactions occur when it is diseased. Disturbances in the body temperature have been observed in a few cases.[3] Cases are on record to support each of these statements, yet it must be admitted that numerous negative cases, that is, cases of tumor of the thalamus in which these symptoms are wanting, throw much doubt on the diagnostic signifi-

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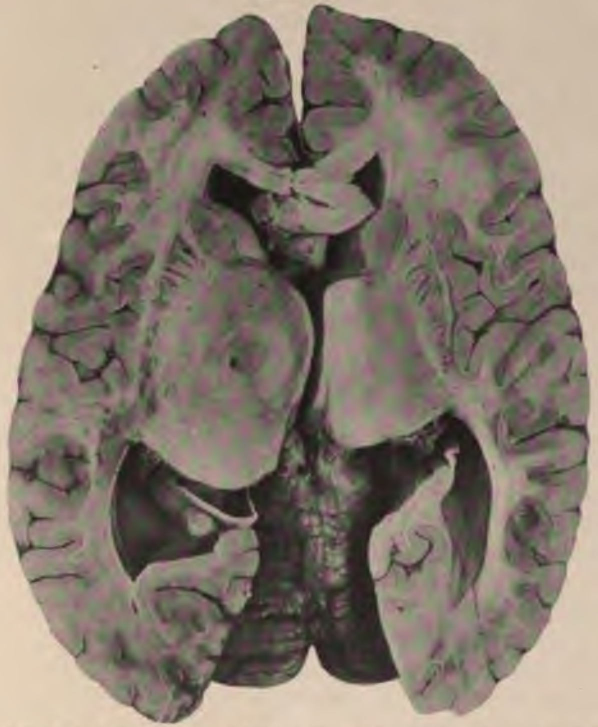
[1] Bruce. Brain, xii. p. 171.

FIG. 240.



Sarcoma of the left optic thalamus and internal capsule. Cyst in the centre of the tumor.

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[1] D. H. Cooke. Lancet, May 28, 1892.

[2] Kirilzew. Neurol. Centralb., 1891, p. 310. Nothnagel, Zeitschrift f. klin. Med., 1889. Bechterew, Neurol. Centralbl., 1884, p. 102.

[3] Cowan. Lancet, December, 1892. Lloyd, Med. News, January, 1892.

cance of the effects mentioned.[1] My own view of the functions of these ganglia is that they preside over many acts which are commonly performed in a purely automatic manner, for example, the posture of the body, the ordinary gait, the facial expression, and probably emotional control; that these acts can, however, be performed voluntarily as well as automatically, and hence, when the automatic mechanism fails, a volitional act takes its place, which practice soon perfects, leaving the individual quite unconscious of his defect, and hence causing few or no symptoms. It is, therefore—on this theory—clear that tumors of the basal ganglia cause few recognizable symptoms.

Tumors of the corpora quadrigemina present symptoms of a focal character which are diagnostic.[2] These consist of a staggering gait similar to that seen in cerebellar disease and symptoms of paralysis or paresis in the muscles controlled by the ocular nerves. The co-ordination in movements of the eyes, the reflex motions of the eyes in following a light or turning toward a sound, and the power of steady gazing at an object are controlled by automatic centres in the corpora quadrigemina. Disease here frequently produces nystagmus and interferes with these automatic movements. The symptoms are not referable to a disease of one third nerve alone, for the pupils are often unaffected. They are often bilateral and more nearly resemble the symptoms of ophthalmoplegia externa. Hence, it is possible to distinguish lesions of the corpora quadrigemina from lesions in the crus cerebri, which cause true third nerve paralysis. The eyeballs are not often equally affected, one being more paralyzed than the other; but neither eyeball, as a rule, is totally paralyzed. The superior and inferior recti are more commonly affected than the lateral muscles. It is the combination of these ocular symptoms with the reeling gait which will lead to the diagnosis of tumor of the corpora quadrigemina when the other (general) symptoms of brain tumor are present.

The focal symptoms of **tumors at the base** of the brain are necessarily very complex. Suffice it to say that tumors lying upon the base of the brain will necessarily compress one or more of the cranial nerves, either upon one side alone or upon both sides, in case the tumor is near the median line. The situation of the tumor can usually be determined by noting the order in which the various cranial nerves are invaded from first to twelfth. (See Figs. 177 to 181, and 198.) The reader is also referred to Chapter XXXIII. for the consideration of lesions at the base, and to Chapter XXXIV. for lesions of the cranial nerves.

Tumor of the crus cerebri is characterized by the combination of hemiplegia of one side with third nerve paralysis of the opposite side. The tumor lies on the side on which the third nerve is affected. To this combination bilateral hemianopsia may be added if the tumor compresses the optic tract.[1]

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[1] Wharton Sinkler. University Med. Mag., October, 1893.

[2] Nothnagel. Brain, xii., 21. Hall, Heidelberg Dissert., 1892.

Tumors in the upper part of the **pons** or in the crus cerebri affect the third and fifth nerves, producing external strabismus, with dilatation of the pupil and ptosis, and also tingling, pain and anæsthesia of the face with ulceration of the cornea, and sometimes grating of the teeth during sleep. Tumors in or near the lower half of the pons involve the sixth and seventh and eighth nerves, causing internal strabismus with contracted pupil, paralysis of the face, including inability to close the eyes, and deafness, with vertigo. Alternating hemiplegia is also produced by a lesion in this region.

Tumors in the medulla may disturb the action of the glossopharyngeal, pneumogastric, spinal accessory, and hypoglossal nerves, producing difficulty in deglutition, irregular respiration, irregular pulse, flushing of the skin, with sweating, sometimes unilateral, projectile vomiting, polyuria, or glycosuria, retraction of the head or rolling of the head upon the pillow, and lastly, paralysis of the tongue, with inability to articulate distinctly or to swallow.

When the tumor lies upon the base of the brain, it not only invades the cranial nerves, but it presses upon the various tracts which traverse the crura, pons, and medulla. Thus it may give rise to hemiplegia or hemiataxia or hemianæsthesia, according to the tract invaded. These symptoms are easily understood by reference to Figs. 177 to 181.

Tumors of the pons, producing pressure upon the middle peduncle of the cerebellum, produce a tendency to stagger in walking. The patient usually staggers toward the side opposite the tumor, but this is not an invariable rule. Tumors of the pons quite uniformly produce a loss of the tendon reflexes at the knee. The control of the bladder and rectum is also frequently impaired.

Tumors of the cerebellum are exceedingly frequent both in children and in adults, and present almost all of the characteristic general symptoms of brain tumor. The situation of the cerebellum held down in a small cavity by the tentorium cerebelli is such that a small growth in the posterior fossa is capable of producing an increase of intracranial pressure quite early in the course of the case. (See Fig. 233.) A tumor in the posterior fossa will also compress the fourth ventricle by displacing its floor or roof, and as a result of such compression an accumulation of fluid in the ventricle, and subsequently in the lateral ventricles, is usual. Hence, symptoms appear earlier in the course of the disease in tumors about the cerebellum than in tumors elsewhere in the brain. Headache, occipital or frontal, is usually the first symptom, and optic neuritis appears very early in the course of the case. The percentage of cases having optic neuritis is much greater in tumors of the cerebellum than in tumors in the cerebral hemispheres.

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[1] Venal. Bullet. de la Soc. Anat., Paris, Jan., 1891.

The focal symptoms of tumors of the cerebellum are not very numerous. They are first staggering in walking, sometimes attended by a strong tendency to stagger or to fall in a particular direction: secondly, symptoms referable to a compression or displacement or stretching of the cranial nerves lying in the posterior fossa.

Tumors in the middle lobe of the cerebellum uniformly produce cerebellar ataxia or a staggering gait. Tumors in the hemispheres of the cerebellum near to and compressing the middle lobe are attended by the same symptoms. So also are tumors which compress the peduncles of the cerebellum, especially the middle peduncles in their course to the pons, or the superior peduncles on their way toward the corpora quadrigemina. Tumors lying within the hemispheres which do not invade these parts do not always produce a staggering gait. Hence, a diagnosis of a tumor of the cerebellum is easy when the tumor lies near to the median line, but is impossible when it lies near the surface in the lateral portion of the hemispheres. Thus in the tumor shown in Fig. 231 there was no staggering; while in the tumor shown in Fig. 232 the patient staggered toward the left side. The early appearance of staggering as related to the general symptoms is, therefore, an important sign in favor of a cerebellar tumor, while the appearance of staggering many months after the development of optic neuritis will merely indicate that a tumor in the hemisphere has finally reached or encroached upon the middle lobe.

The early appearance of cranial nerve symptoms, such as paralysis of the face, deafness, disturbance in swallowing, symptoms referable to the vagus nerves or paralysis of one-half of the tongue, indicates that the tumor lies near to the medulla and pons, that is, upon the inferior surface of the cerebellum. The appearance of these symptoms late in the disease, long after the development of optic neuritis, will indicate that the growth has finally reached the inferior surface, or more probably that its size is so great as to have displaced the cerebral axis. Such a tumor is shown in Fig. 232.

The characteristics of cerebellar ataxia are a staggering gait with steps of irregular length and position, the body swaying like that of a person intoxicated. The ataxia of the legs and of the body is attended by a decided sense of vertigo, and does not usually persist when the patient lies down. These characteristics, together with the fact that the knee-jerks are exaggerated in cerebellar tumor, will enable anyone to distinguish this disturbance of gait from that appearing in locomotor ataxia. Cerebellar ataxia is due to a disturbance in the mechanism of equilibrium in so far as this depends upon impressions coming to the brain from the muscles of the trunk and legs. When the staggering is in one direction only it is an indication that the middle peduncle of the cerebellum is invaded by the tumor. This unilateral staggering is not a common symptom. In four-fifths of the cases recorded the patient has staggered away from the side on which the tumor lies: in the remaining fifth the patient has staggered toward the tumor. The direction of staggering is not, therefore, an absolute sign of the side on which the tumor lies. When the middle peduncle of the cerebellum is invaded there are usually cranial nerve symptoms produced, and these are always upon the side on which the tumor lies. Hence the combination of the staggering toward one side with cranial nerve symptoms of the opposite side will indicate inevitably which peduncle of the cerebellum is involved. Thus in the case shown in Fig. 232 the diagnosis was made before death from this combination of symptoms. When the superior peduncles of the cerebellum are invaded, nystagmus, paralysis of the oculomotor muscles, and blindness are developed. In fact it is impossible to differentiate a tumor invading the superior peduncles of the cerebellum from a tumor of the corpora quadrigemina.

Diagnosis. The review of the symptoms just described will convince the reader that in the majority of cases of tumor of the brain there are sufficient evidences of the existence of disease within the cranial cavity, and that the gradual development and progress of the disease will enable the physician to come to the conclusion that it is of the nature of a tumor. If in any case these symptoms which are present are carefully classified, the general symptoms being separated from the local symptoms, and the order of appearance of the local symptoms fully determined, it will usually be possible to reach a conclusion as to the situation of the tumor. The combination of local symptoms is sometimes as characteristic as their order of appearance and method of extension, as, for example, in cases of tumors of the crus or of the corpora quadrigemina.

It is not to be forgotten, however, that tumors of the frontal lobes and tumors of the temporal lobes, especially those situated in the right hemisphere, often fail to cause any local symptoms. The absence of distinct local symptoms, therefore, will point to these localities as the probable position of the suspected tumor.

It is not to be forgotten that many local symptoms are produced indirectly by pressure upon parts not far from the tumor, but yet not directly invaded in its growth.

It is also to be remembered that tumors pressing upon large vessels of the brain may so disturb the circulation as to produce symptoms quite similar to those of thrombosis, and these symptoms may be due to suspension of function of a part lying at some distance from the tumor.

The diagnosis of the variety of the tumor present can be reached only by careful study of the general history of the patient and by a consideration of those facts that have been already mentioned in the discussion of the varieties of brain tumor.

It is therefore evident that in the diagnosis of the disease there are always three questions to be settled: first, the existence of a tumor; secondly, its situation; thirdly, its variety. A study of the symptoms will usually enable the physician to reach an answer to

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It is therefore evident that in the diagnosis of the disease there are always three questions to be settled: first, the existence of a tumor; secondly, its situation; thirdly, its variety. A study of the symptoms will usually enable the physician to reach an answer to the first two questions, but the answer to the last question will always remain uncertain.

The symptoms of brain abscess may be the same in kind as those of brain tumor, since both produce an increase of intracranial pressure and a progressive destruction of brain tissue. In their origin, mode of development, progress, and termination, however, there are not infrequently marked differences. Brain abscess develops most commonly after severe injuries or in conjunction with suppurative affections of the inner ear and of the nasal and orbital cavities, and with caries of any of the cranial bones which lie in contact with the membranes. These rarely occur prior to the development of brain tumor. The symptoms common to tumor and abscess may develop after a blow on the head, but when the condition is one of abscess the symptoms appear in more rapid succession, with greater severity, and more frequently with fever than when the condition is that of tumor. Furthermore, the symptoms of abscess, after appearing suddenly, often subside, the abscess becoming latent, and all symptoms disappearing for months or years, and then breaking out again with suddenly fatal termination. This course contrasts markedly with that in a case of a tumor where a gradual progress with slowly increasing intensity of all the symptoms is found. The constant addition of new symptoms is usual in tumors, and a temporary remission rather than intermission of the symptoms is the rule when the progress is not continuous. There may also be some points of distinction found in the individual symptoms. Thus headache is more severe and paroxysmal with tumor; optic neuritis is much more commonly found with tumor; mental changes are more gradual and constant with tumor, and local symptoms are slower in onset and more apt to develop with tumor. A complication of tumor not infrequent is cerebral hemorrhage. Meningitis is the usual complication of abscess. Lastly, a duration of from one to two years, with symptoms constantly present, points directly to tumor.

Tubercular meningitis is under certain circumstances easily mistaken for cerebral tumor. This is not true of the ordinary cases of tubercular meningitis with acute hydrocephalus, which develop marked symptoms rapidly and terminate fatally within four or six weeks. But there are a number of cases of tubercular meningitis which present a chronic course with gradual progress, and in which the diagnosis from tubercular tumor is almost impossible. It is true that the symptoms often develop rapidly in these cases, and yet this is sometimes apparently the case in tumor, for, unless a patient is carefully watched, the early symptoms of tumor may escape notice for some time. The symptoms of chronic tubercular meningitis may be the same as those described as general symptoms of brain tumor, but the headache is more severe in meningitis and more continuous; there is more likely to be hypersensitiveness to light, sound, or touch in meningitis, and optic neuritis develops less frequently, less rapidly, and with less intensity than in tumor. Tubercles upon the choroid are found more frequently in meningitis than in tubercular tumor. It is, of course, understood that a localized meningitis may give rise to the same symptoms as a small tumor, and then the differentiation is impossible. This is more common about the base of the brain, in the region of the cranial nerves, than elsewhere. It is also to be remembered that a chronic progressive meningitis may develop in the vicinity of a tumor. Here, again, the diagnosis will be impossible. A gradual subsidence of the symptoms, with recovery, will point to meningitis rather than to tumor.

Chronic hydrocephalus, while not infrequently the result of tumor or meningitis, may be due to a chronic inflammation of the ependyma of the ventricles. It then advances slowly. The fluid within the ventricles produces pressure upon the brain, causing atrophy. The course is chronic, and the general symptoms are those of cerebral tumor. The local symptoms, however, differ in some respects from those of tumor. Spastic paralysis develops with chronic hydrocephalus without localized spasms, and is always bilateral; the lower limbs are affected more intensely than the upper. The child presents the extended, adducted, stiff legs, with overlapping knees, rigid muscles, increased tendon reflexes, and the spastic gait so familiar in infantile diplegia (see Chapter XXVII.), and, in addition, the hands move without proper co-ordination. As the disease progresses the crura cerebri and pons may be displaced by the pressure of the fluid, and irregular symptoms due to stretching of the cranial nerves may appear. These, with the paraplegia, may lead to a suspicion of a tumor of the pons or base of the brain, and only by the order of development of the symptoms can the differentiation be made.

The diagnosis between cerebral tumor and cerebral hemorrhage will be necessary only in a few cases in which the onset of the symptoms has been very sudden. There are a few cases of tumor, chiefly glioma, in which the growth has been latent for some time, and has then given rise suddenly, after a blow on the head, or exposure to the sun, or some other accidental influence, to well-marked symptoms. These are usually both general and local symptoms, chiefly the latter. The suspicion of a tumor will be aroused if, after such an apoplectic stroke, the symptoms persist and increase instead of subsiding, and if headache, convulsions, and optic neuritis appear. Hemorrhage alone never gives rise to the last-named symptom.

There are a few general diseases which present certain symptoms somewhat similar to those occurring in brain tumor, and these should always be kept in mind. They are extreme anæmia, with defective vision, from myopia or hypermetropia; chronic lead poisoning and chronic diffuse nephritis or contracted kidneys. The knowledge that these diseases may simulate brain tumor will lead the physician to be upon his guard. It is not necessary to mention the numerous points of the differential diagnosis which will enable a definite conclusion to be reached in any case.

The Course of the Disease. The general history of the progress of a patient suffering from brain tumor has to some extent been indicated in the discussion of the symptoms. Suffice it to say that a gradual increase in the number and intensity of the various symptoms is usual. In some cases the general symptoms precede the local symptoms by several months, and optic neuritis does not often appear within three months of the beginning of the symptoms, unless the tumor is in the cerebellum or on the base of the brain. In some cases the local symptoms appear before the general symptoms, especially if the tumor is located in the motor cortex, when the case may be regarded for some time as one of cortical epilepsy until the general symptoms of brain tumor supervene.[1]

As the case goes on and both local and general symptoms become more numerous the suffering of the patient becomes more intense.

If the case is one of gumma it may be possible to relieve the symptoms and, by a progressive course of treatment, to cure the patient entirely. Under these circumstances the symptoms gradually subside and become less in intensity up to recovery. In other cases it is possible to locate the tumor absolutely in a position accessible to the surgeon and to remove it by operation, and under these circumstances the recovery of the patient is gradual but progressive after the operation is over. The brain resumes its functions after the pressure of the tumor is removed. When a portion of the brain has been injured in the removal of the tumor recovery may be imperfect, with some defects of sight, or motion, or sensation remaining. In those cases that are not subject to specific treatment and in which the tumor cannot be removed the course is progressively downward, the patient suffering more and more intensely as the tumor grows, and finally passing into a state of coma or dying in convulsions.

The average duration of the symptoms is said to be three years, but individual cases vary greatly.

There are a few cases, however, in which, either spontaneously or under specific treatment, a tumor has ceased to grow and the patient has apparently recovered and remained quite well for some months or even years, the brain apparently resuming its function. Two such cases have come under my observation. In one the recovery lasted four months, sudden death following, due to the rupture of a cyst which lay at the side of a sarcoma. In the other an interval of eight years occurred, after which the symptoms returned and caused death. In the last case optic neuritis, which was present at the first attack, subsided entirely, but recurred at the second attack. The tumor was a sarcoma in the cerebellum. In both cases there was no evidence of syphilis, yet the treatment, which was apparently successful, was by inunctions of mercury and large doses (300 to 400 grains daily) of iodide of potassium.

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[1] Kocher. Zeitsch. f. Chirurgie, June, 1893.

In some cases of tumor of the brain sudden death has occurred unexpectedly. I have witnessed four such deaths in patients about to be subjected to operation. In two of these cases tumor of the cerebellum near to and pressing upon the medulla was found. In one of these cases the heart continued to beat six hours after voluntary respiratory movements had ceased.

Prognosis. It is evident from this statement of the course of the disease that the general prognosis in brain tumor is unfavorable. We have seen from a table on page 585 that but 9 per cent. of tumors of the brain are open to operation. It is therefore evident that in the vast majority of the cases we cannot give the patient any hope.

There is always a possibility that a tumor may be syphilitic. It is always imperative, therefore, as soon as the diagnosis is established, to try the effect of specific treatment. My preference is for the use of inunctions of mercury, one drachm of blue ointment being rubbed in at night after a hot bath, a different part of the body being selected for each application, and after the application being covered with bandages so that the process of absorption of the ointment remaining upon the skin may occur during the night. At least two ounces of blue ointment should thus be used, and if improvement is evident the inunctions may be repeated after an interval of two weeks. The occurrence of salivation will necessitate the cessation of the use of mercury temporarily. At the same time large doses of iodide of potash should be given, beginning with twenty grains three times a day, and increasing the dose one or more grains daily until 100 grains three times a day have been reached. The iodide may be given in Vichy water or in milk, before or after meals, according to the condition of digestion. My preference is to give it before meals.

Care in the regulation of the diet, the use of simple and nutritious food, frequent massage to aid digestion, and a daily hot bath at a temperature of 100° F. continued for fifteen to twenty minutes are essential during the taking of these large doses of iodide.

When the tumor is of a gummatous nature a decided improvement in all the symptoms, and particularly in insomnia and headache, should be observed within a month. Such improvement may, however, occur in cases of either sarcoma, or glioma, or cystic tumors. It is therefore necessary to keep up this treatment for two months longer. If the improvement continues and the patient gradually recovers, it is probable that a gumma has been absorbed. In a case recently under my observation in which the symptoms in January, 1893, were intense headache, insomnia, mental apathy, staggering gait, great general weakness, optic neuritis in both eyes, with partial blindness in the right eye and total paralysis of the right third nerve, partial anæsthesia of the right side of the face, and paresis of the right sixth and seventh nerves, there was by November, 1893, a complete recovery, which still persists, an occasional nocturnal headache being the only symptom remaining. Five courses of inunction have been employed and iodide has been given to the extent of 250 grains a day, the dose being varied from time to time when symptoms of intolerance appeared. Thus in ten months a basal gumma of considerable size has been absorbed by persistent treatment.

If the tumor present is a sarcoma or glioma, and an improvement has occurred during the first months of treatment, such improvement will not always persist, and therefore a return of the symptoms during the course of specific treatment is a pretty sure proof that the tumor is not of the nature of a gumma. It is useless to continue specific treatment after three months of thorough trial; it is better to refuse medical treatment or to depend entirely upon palliatives under these circumstances. It must be stated, however, that some authorities believe that tumors of the brain, not syphilitic in nature, have been caused by the persistent use of large doses of iodide. In every case, therefore, where surgical treatment is impossible it is well to employ this remedy.

Treatment. 1. **Medical Treatment.** Headache can be relieved by a free use of phenacetin, antipyrine, or acetanilid. The doses of these drugs which must be used are larger than those commonly employed, and it is my practice to begin with the ordinary dose and rapidly increase the number of doses given, combining with the drug any heart stimulant, caffeine being the one preferred. If the patient be carefully watched while this is being done it will soon be found possible to give safely twenty grains of antipyrine, fifteen to twenty grains of phenacetin, or ten grains of acetanilid at a dose, and this dose may be repeated after three hours, provided the headache returns. In a few cases the headache may be benefited by ice-bags to the head, by hot baths, or by ergot. If these remedies are useless resort must be had to morphine, but this drug is especially unsatisfactory in the treatment of headache from brain tumors, unless very large doses are given. Vomiting and vertigo in brain tumor are best relieved by the use of bromide of sodium or by hydrobromate of hyoscine in $\frac{1}{100}$ gr. dose repeated every four hours.

The course of optic neuritis cannot be arrested, though it may be somewhat delayed by cupping the temples. While strychnine will at any time, when given hypodermically, improve temporarily the power of vision, yet it does no permanent good, and is thought by some to hasten the progress of the disease.

Epileptiform convulsions may be reduced in frequency by the free use of bromides, but cannot be arrested as long as the disease goes on. It is thus evident that the treatment of the general symptoms of brain tumor is exceedingly unsatisfactory. There is no treatment known that will in any way affect the local symptoms, massage and electricity to paralyzed limbs being mere palliatives, capable only of maintaining the nutrition of the muscles.

2. **The surgical treatment** of brain tumors is a subject which has awakened much interest of late, inasmuch as over 400 tumors have been operated upon within the past twelve years.

I have made a collection of 377 tumors of the brain in which an operation has been attempted.[1] The results are shown in the following table:

TABLE OF RESULTS OF OPERATIONS FOR BRAIN TUMOR.

	Cerebral.	*Cerebellar.*
Cases in which the tumor was found and removed and patient recovered	154	16
" " " " " " " " died .	52	8
" " " " but could not be removed . .	22	12
" " " " not found	91	22

It is evident that in this disease, formerly considered absolutely fatal, a certain proportion of patients can be saved. Such favorable results make it imperative to consider the possibility of operation in every case of brain tumor. Success in these operations depends (1) on the situation of the tumor; (2) on its removal early in the course of the disease; (3) on the variety of tumor found; (4) on the amount of brain tissue destroyed either by the tumor or in the course of the operation.

An analysis of the successful cases shows that they were chiefly tumors situated about the fissures of Rolando or Sylvius, giving rise to motor or aphasic symptoms easily localized. I have had a number of these cases; in two perfect recovery has been the result, and the patients are now alive and well. A number of tumors have been successfully removed from the frontal convolutions, from the parietal region, and from the occipital region, but the percentage of failures in these localities, due probably to uncertainty in diagnosis, was greater than in the motor region. I have had successful cases in each of these localities. The cerebellum is very difficult to reach, only a small part of each hemisphere being accessible through the occipital bone, and hence the successful cases of removal of cerebellar tumors are small in number. In several patients with cerebellar tumor whom I have had trephined it has been impossible to reach or to completely remove the tumor. In any case, therefore, where the tumor is cortical, can be localized accurately, and is accessible, the chance of success is good. In some cases tumors beneath the cortex can be removed. It is quite safe to incise the brain to a depth of several inches if the ventricle is not opened. If a tumor is not seen at an operation it may be felt for, the finger being thrust into the brain at the bottom of a sulcus. Thus in a case of deep lying tumor of the parieto-occipital region, removed for me by McCosh, it was only at the second operation and by a deep incision that this was found. It proved to be an encapsulated sarcoma with a pedicle attached to the falx, and its removal was followed by apparent recovery, hemianopsia remaining. The tumor, however, recurred some months later, and the patient died. It is to be remembered that deep incisions or lacerations of the brain in the removal lead to permanent symptoms, and the scar is likely to prove the focus of irritation for subsequent epileptic attacks.

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[1] Brain Surgery. Wm. Wood & Co., 1893. New York Medical Record, February 1, 1896. Montreal Medical Journal, November, 1897. Journ. of Nerv. and Ment. Dis., June, 1903, where each case is tabulated.

The second factor in success is the promptness with which the need of an operation is recognized and the procedure undertaken. Many patients could have been saved, who have died, had the tumor been removed at an earlier time in the course of the case. The moment an accessible tumor is diagnosticated an operation should be undertaken. It is a mistake to try medical treatment, even antisyphilitic treatment in syphilitic cases, as the operation even in these affords rapid relief, and in cases where it fails valuable time is lost.

The third factor in success is the variety of tumor found. The following table illustrates this fact:

TABLE OF VARIETY OF TUMOR REMOVED, AND RESULT.

Variety.	*Recovered.*	*Died.*
Sarcoma	52	20
Gliosarcoma	10	0
Cyst	29	11
Angioma	7	1
Gumma	8	3
Tubercular	19	8
Fibroma	7	2
Endothelioma	5	4
Glioma	15	13

Sarcoma, gliosarcoma, and fibroma are usually hard, encapsulated, easily separable from the brain tissue, and can be removed without laceration.

Cysts of the brain are easily emptied when found, but the mere abstraction of the fluid gives no permanent relief, as it reaccumulates. It is necessary, therefore, either to drain the cyst and thus secure its closure from the bottom by the approximation of its walls, or else to remove the whole of the cyst by dissecting it out from the brain. The latter is the best method when the cyst is on the surface, but is a dangerous process, and is sure to be followed by the formation of scar tissue, which in itself is a constant irritant to the brain. Drainage is necessary in deeper lying cysts. If, however, the cyst is a part of a glioma the removal of its wall and adjacent tissue affords less hope.

Angioma, if lying on the cortex and if made up, as is usual, of vessels from the pia, can be easily removed if care be taken to ligate all the vessels entering the tumor before any are cut. But such ligation may be followed, as in one patient of my own, by extensive softening of the area the veins of which were tied.

Gumma, if hard, is easily taken out, but if soft and infiltrating the pia is more difficult of removal. The hard type requires surgical treatment; hence it should be removed. The soft type will yield to medical treatment, which should in any case follow the operation when a gumma is found.

The question of operative interference when a tubercular tumor is diagnosticated has given rise to some discussion, von Bergmann being opposed to such operations and Horsley being in their favor. It must be admitted that a permanent cure by operation is less likely to be achieved in the case of a tubercular tumor than in the case of a non-tubercular tumor, there being not only the danger of recurrence but also the possibility of the existence of undetected tumors elsewhere in the brain and the danger of the development of tubercular meningitis subsequently to the operation. An operation is capable of prolonging life, and hence should not be wholly condemned.

The prospect of recovery when an endothelioma or a glioma is found is not good, for it is rarely possible to remove these tumors completely, as they infiltrate the brain, and even if removed they are liable to recur. Carcinoma is particularly unfavorable because it is usually secondary to carcinoma elsewhere.

The degree of ultimate recovery after such operations will depend upon the amount of destruction of brain tissue produced by the tumor or by the necessary incisions of the operator. Sometimes this is considerable, and the patients are left incapacitated for life. In many cases, however, of which I can record several, a complete recovery has occurred, even when extensive paralysis had preceded the operation. In the table of results the word "recovered" indicates that the patient survived the operation and was improved by it. It is not to be interpreted as implying a complete restoration to health, which rarely occurs. It is no small result in such cases to save a patient's life and to arrest the progress of a fatal disease.

The surgical details of the operation do not require description, being beyond the scope of the physician.[1] They are many and complex. The operation is not to be undertaken lightly or without experience, and it is better for the physician to refer such cases to some surgeon who is familiar with the methods of procedure.

When an operation for a brain tumor is advised it should be explained that it is an exploratory operation, that it may save life, but that it may leave disabilities which will be permanent; that it may fail because of the impossibility of removal of the tumor, either because of its deep position or of its nature. At the same time it should be urged as affording a possible cure for an otherwise fatal disease.

Horsley has advised that in cases in which the tumor is inaccessible a large opening be made in the skull and that a drainage tube be passed within the dura, or that the ventricle be drained to relieve, if possible, the intracranial pressure. I have seen this done, but it seems to me of problematical value, as it merely prolongs the misery in a hopeless condition.

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[1] See Brain Surgery, by M. Allen Starr, M.D. Wm. Wood & Co., 1893. Also Brain Surgery, by W. W. Keen, M.D. Reference Handbook Med. Sciences, Wm. Wood & Co.

CHAPTER XXXIII.

BULBAR PARALYSIS.

THE CEREBRAL AXIS.

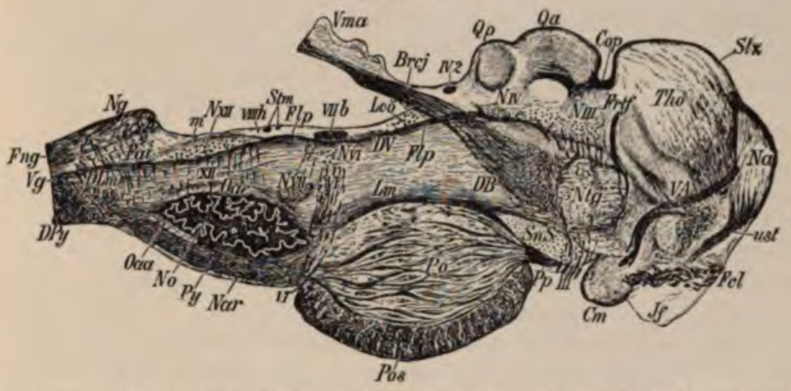
THE cerebral axis consists of the crura cerebri, pons Varolii, and medulla oblongata, which lie upon the base of the skull. (See Fig. 199, page 476.) It contains the nuclei of origin of all the cranial nerves except the first and second. These are arranged in groups, lying upon the floor of the fourth ventricle or near to it. It also contains the great motor and sensory tracts connecting the cerebrum and cerebellum with each other and with the spinal cord. Fig. 241 shows a lateral view of the cerebral axis and Fig. 198 (page 475) shows a transparent diagram which indicates the position and relative arrangement of the nuclei within it. Figs. 177 to 182 (pages 440 to 446) show the various tracts passing through the cerebral axis.

From a study of these diagrams and from a consideration of the symptoms of lesions in the cerebral axis already discussed, pages 439 and 474, it is evident that gross lesions such as hemorrhages, small areas of softening due to thrombi or emboli, plaques of sclerosis, and tumors of the cerebral axis, can be easily located by their effects upon the cranial nerves and upon the sensory and motor tracts. The characteristic features of such lesions are alternating paralysis or anæsthesia when the lesion is unilateral, and paraplegia with cranial nerve symptoms when it is bilateral. The small size of the cerebral axis and the importance of the tenth nerve centres to life make it evident that lesions which are larger than half an inch in diameter are usually immediately fatal. If, however, a lesion spares the tenth nerve nuclei, the patient may live, and then a diagnosis of bulbar lesion is not difficult. The prognosis is, however, more serious than in tumor, hemorrhage, or softening elsewhere in the brain, as an extension of the lesion may end life at any time, and regeneration in these nuclei and tracts does not occur.

BULBAR PARALYSIS.

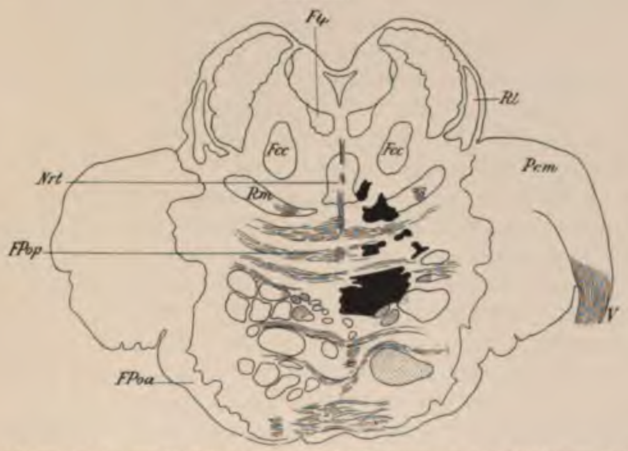
Any affection which involves the cerebral axis may be termed bulbar paralysis. Thus cases of hemorrhage, which are rare, or of softening, which are common in the crura, pons, or medulla, may be described as "acute apoplectiform bulbar paralysis." But since such cases differ from cases of cerebral apoplexy in no respect excepting in the peculiar combination of their symptoms, due to the location of the lesion, it seems unwise to establish a special category of diseases for these cases. The facts already stated in regard to the pathology of apoplexy (page 489), and in regard to its symptoms,

FIG. 241.



The cerebral axis. The cranial nerves and their nuclei are numbered *III* to *XII*. *Brcj*. Superior peduncle of cerebellum. *Cm*. Corpus mammillare. *Cop*. Posterior commissure. *DB*. Decussation of superior peduncle. *DLm*. Sensory decussation in medulla. *DPy*. Motor decussation in medulla. *DV*. Crossed bundle of V. nerve. *Fai*. Internal arcuate fibres. *Fcl*. Fornix. *Fng*. Funiculus gracilis. *Flp*. Posterior longitudinal bundle. *Frtf*. Fasciculus retroflexus. *If*. Infundibulum. *Lco*. Locus cæruleus. *Lm*. Lemniscus. *Na*. Anterior nucleus of thalamus. *Nar*. Nucleus arcuatus. *Ng*. Nucleus gracilis. *No*. Olive. *Ntg*. Nucleus tegmenti. *N*. Red nucleus. *Oaa*. Ventral olivary nucleus. *Oae*. Dorsal olivary nucleus. *Po*. Pons. *Pos*. Superficial transverse fibres of pons. *Pp*. Pes pedunculi. *Py*. Pyramid. *Qa*, *Qp*. Corpora quadrigemina. *Sn*. Substantia nigra. *Stm*. Striæ acusticæ. *Stz*. Stratum zonale of the thalamus. *Tho*. Thalamus. *Ust*. Lower thalamic bundle. *Va*. Vicq d'Azyr's bundle. *Vg*. Anterior column. *Vma*. Valve of Vieussens. (Obersteiner.)

FIG. 242.



Section through the pons Varolii, showing multiple hemorrhages which caused death. (Comte des paralysies Pseudo-bulbaires, Paris, 1900.)

and especially those produced by lesions in this region (page 474) apply to this condition and make it unnecessary to do more than mention an affection which, though extremely rare, has received some attention in recent text-books. Fig. 242 shows a diagram of such a lesion, a number of small hemorrhages in the pons Varolii having caused the death of the patient. Acute bulbar paralysis has been already described in the chapter upon encephalitis; it being also known as polio-encephalitis inferior. (See page 554.)

By bulbar paralysis a disease of a chronic progressive type is usually indicated, also called glosso-labio-laryngeal paralysis. This disease is exactly homologous to chronic anterior poliomyelitis, the lesion being in the cerebral axis and not in the spinal cord. It is a slowly advancing degeneration in the nuclei of origin of the motor cranial nerves, one after another being involved, usually from below upward, but occasionally from above downward. In some cases it is a part of a more widespread affection, viz., amyotrophic lateral sclerosis, and many cases which present the typical features of bulbar palsy, if they survive, develop the additional symptoms which characterize that disease. (See page 237.) But in many cases the bulbar symptoms are the only ones to present themselves, and produce a very typical picture which is easily recognized.

Etiology. The etiology of bulbar paralysis is obscure. In a few cases it appears to have been a congenital affection, and as, in these it has occurred in two or more children of a family whose parents were related to one another or in children who showed signs of degeneration, it has been considered an hereditary or a family disease and due to defective development of the brain. In one such case hereditary syphilis was the manifest cause. The majority of the cases develop between the thirtieth and fiftieth years of life, though no age is exempt. Falls and blows on the head, emotional strain, and cold have been thought to be causes of the disease. Overwork of the facial muscles, such as occurs in players on wind instruments, has been thought to be a cause.

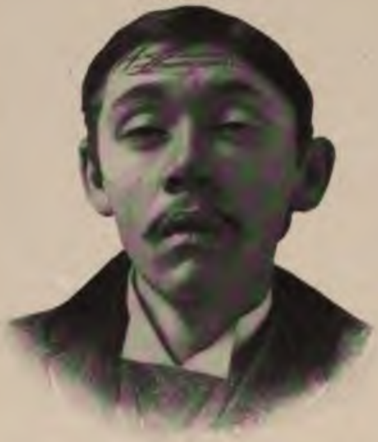
Pathology. The pathology is a progressive atrophy of the motor neurones in the hypoglossal, glossopharyngeal, spinal accessory, pneumogastric, facial, and motor part of the trigeminal nerves and occasionally of the abducens and oculomotor nuclei also. The bodies of the neurones undergo a progressive atrophy, lose their branches, both dendrites and axones, become smaller, and finally are reduced to small nuclei or disappear wholly. (See Plate III.) The axones degenerate in the same manner and leave an empty sheath. The muscles to which they go atrophy. The pathology of the disease is therefore identical with that of chronic anterior poliomyelitis (*q. v.*). This lesion may be limited to the gray nuclei of the cerebral axis. But in some cases sclerosis of the pyramidal (motor) tracts in the medulla and pons has been found, and this in a few cases could be traced into the lateral and anterior median columns of the spinal cord. In such cases it is evident that the disease was amyotrophic lateral sclerosis and that the bulbar paralysis was only the first stage of that affection.

Symptoms. The symptoms of bulbar paralysis usually begin in the tongue, causing a slight defect in articulation, words which contain the linguals, d, l, n, r, s, t, being badly pronounced. As the disease goes on this difficulty of speech is increased by the development of paralysis in the pharynx, which makes the speech nasal, and of paralysis of the lips, which prevents the enunciation of b, p, v, w, y, and finally it is almost impossible to understand these patients, the vowel sounds alone being clearly produced. When the larynx itself becomes paralyzed even phonation is indistinct. This symptom has been termed dysarthria to distinguish it from aphasia. The paralysis of the tongue is attended by fibrillary twitching and by atrophy, which throws the mucous membrane into folds. A reaction of degeneration may be found in the muscles of the tongue.

While the symptoms are developing disturbance in the act of chewing and of swallowing appears. The food cannot be rolled about in the mouth on account of the paralysis of the tongue and cannot be carried backward to the pharynx. The pharyngeal muscles are involved, and hence their automatic action is imperfect; hence the food is not grasped and propelled into the œsophagus, but may stick in the throat and get into the larynx. The automatic closure of the epiglottis on arrival of food near it is also impaired by paralysis of the laryngeal muscles; hence fluids trickle into the larynx and fits of choking become frequent. These patients sometimes attempt to aid themselves by pushing the food back into the pharynx with the finger or by lying down when they attempt to swallow fluids. The paralysis of the pharynx and uvula may lead to regurgitation of fluids through the nose. When the paralysis reaches the muscles of mastication chewing is impossible, and semisolid food only can be taken. It is probably one reason for the rapid emaciation of these patients that the difficulty of eating deprives them of a proper amount of nourishment. The difficulty in deglutition forms one of the chief dangers of the disease, for foreign bodies get into the larynx and lungs and choke the patients or set up pneumonia, which is fatal. For this reason feeding by the stomach-tube should be begun early, and the patient should be instructed how to pass the tube, and thus should avoid all acts of swallowing.

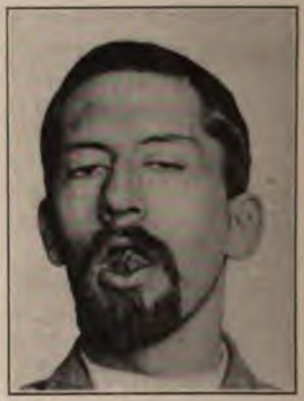
A further set of symptoms is referable to the facial muscles. The lower muscles become weak and atrophy, causing a lack of facial expression and a flattening of the face from obliteration of its folds. The muscles about the mouth are relaxed and the mouth cannot be firmly closed, and the lower lip hangs down. Hence the saliva runs out, and the patient holds a handkerchief to catch the drooling. He can no longer whistle or blow. The jaw-jerk may be increased. The muscles closing the eye are very rarely paralyzed. The lower muscles of the face and of the tongue are in a state of constant fibrillary contraction causing a fine tremulous, wavy movement of the skin and mucous membrane. As the facial muscles atrophy a partial reaction of degeneration can be obtained. The paralysis of the muscles of mastication leads to an inability to close the jaw, which makes it necessary for the patient to support it by the hand. The tongue in its final state of paralysis lies on the floor of the mouth, cannot be put out or moved about, the soft palate hangs motionless and does not rise in speaking or in swallowing; hence fluids return through the nose. In some cases the oculomotor nuclei become affected and various forms of strabismus and double ptosis develop. Thus in the patients shown in Figs. 243 and 244 there was not only a paralysis of the facial muscles and of the tongue, but double ptosis with paralysis of all the muscles of the eyeballs.

FIG. 243.



Patient suffering from bulbar paralysis. Double ptosis, slight external strabismus, facial paralysis with lack of facial expression and inability to close the lips are to be seen. The effort to open the eyes causes a wrinkling of the forehead.

FIG. 244.



Bulbar paralysis. Double ptosis, with external strabismus, flattening of the face, inability to close the mouth, and atrophy of the right half of the tongue are to be seen. (Icon. de la Salpêtrière.)

Finally, the respiratory movements of the larynx are interfered with, a peculiar stridor is heard in respiration, the breathing becomes rapid, and death by asphyxiation occurs. In the early stage a laryngoscopic examination shows weakness of the adductors, which later on becomes a total paralysis. In a few cases the oculomotor nerves and the abducens have been paralyzed, causing ptosis, and strabismus with all the characteristics of ophthalmoplegia (*q. v.*).

There is never any pain or anæsthesia of the face, but headache in the occiput or pain in the nape of the neck is sometimes complained of. Sometimes an increase of knee-jerks is present.

A very constant symptom of bulbar paralysis is an emotional state which leads to undue laughter or more often to frequent crying without apparent cause. This, as already stated, must be regarded as a local symptom of pons disease. In a few cases symptoms of spastic paraplegia appear and the disease goes on to amyotrophic lateral sclerosis. In other cases an atrophic paralysis develops in the hands and arms, and it is evident that a chronic anterior poliomyelitis has followed the bulbar palsy. It has already been stated that in both of these diseases bulbar paralysis may develop as a final result. These facts point to a common origin and pathology in all three affections, which may, therefore, be regarded as stages or varieties of onset in one disease.

The course of bulbar paralysis is slowly progressive. In a few cases there may be for a time an arrest of symptoms, but they soon reappear, and the result is uniformly fatal. The average duration of the disease is two years, but I have seen several rapid cases which terminated within a year of the onset. Death is due to choking, to pneumonia or bronchitis set up by inhaling food, or to starvation.

Diagnosis. The diagnosis of bulbar paralysis presents no difficulty. It is to be remembered, however, that similar symptoms are caused by tumors of the cerebral axis, though these are rarely so located as to produce bilateral symmetrical symptoms. I have seen one case, however, in which the entire absence of general symptoms of brain tumor and the slowly progressive development of bulbar paralysis led to this error, which was only revealed at the autopsy. The history of the slow progress of the symptoms will serve to distinguish bulbar paralysis from vascular lesions in the cerebral axis. But cases of aneurism of the basilar artery sometimes present very similar symptoms, though in the few cases thus far recorded spastic paraplegia has appeared early.

The occurrence of spinal and cortical symptoms will distinguish multiple sclerosis from this disease. In the course of multiple neuritis either of diphtheritic or of toxic origin cranial nerve symptoms somewhat like those of bulbar palsy may occur. But the history of the case, its rapid progress, the occurrence of other symptoms referable to the nerves of the extremities, and the rapid recovery leave no doubt as to the diagnosis from bulbar paralysis.

There is a condition quite like that of bulbar paralysis which is produced by bilateral lesions in the facial motor area of the cerebral cortex lying in the operculum or in the motor tracts from those areas to the nuclei of the cranial nerves, especially in the internal capsule near the lenticular nucleus and in the lenticular loop. This condition is naturally a very rare one, but it has been observed. It may be a congenital defect with porencephalus, and then the symptoms appear in infancy. It may be due to multiple sclerosis or to several separate attacks of an apoplectic nature, each causing unilateral symptoms, but the final result being a symptom-complex resembling bulbar palsy. The history of the development of the symptoms should prevent any mistake of diagnosis. And in these cases the paralysis is not accompanied by rapidly advancing atrophy, fibrillary tremor, and reaction of degeneration, which can be demonstrated in cases of bulbar paralysis. There is usually a weakening of the mind, a dementia, and not uncommonly some form of aphasia. And in these cases evidences of arterial disease aid the diagnosis as well as the non-progressive course of the symptoms.

Finally, it is necessary to differentiate bulbar paralysis from a disease known as asthenic paralysis or myasthenia gravis. This was first described by Erb, later by Oppenheim, and then by Eisenlohr, Hoppe, and others.[1] These authors related cases of supposed bulbar paralysis in which either no lesion was found after death, or in which unexpected recovery ensued. The symptoms were much like those of bulbar paralysis, though ptosis, paralysis of the orbicularis palpebrarum, and weakness of the muscles of the neck, which are very rare in that disease, appeared early in these cases. This gives a peculiar and typical facial expression. (See Fig. 243.) Furthermore, in these cases a great general muscular lassitude and undue fatigue on exertion were quite constant, so that a few movements exhausted the patient completely, rendering him incapable of walking or of using his hands. Even the electrical excitability of the muscles diminishes progressively as the current is applied, until no contraction can be produced, yet after a period of rest it is found to have returned. This peculiarity, termed electrical fatigue of a muscle, is characteristic of the disease, as shown by Jolly and Murri. The muscles which are affected do not atrophy and show no reaction of degeneration. The course of the disease differs from that in bulbar palsy, for while the onset may be similarly slow and the various symptoms may all be alike in bulbar and asthenic paralysis, the disease is not steadily progressive. Periods of improvement or even entire subsidence of the symptoms occur. The patient may recover and be free from all signs of disease for some months, though the recovery is never permanent, as almost all cases die within a few years. And if the symptoms prove fatal no lesion in the nervous system has been found. Many theories for this disease have been offered, but none is wholly satisfactory. The toxic theory seems most probable, viz., that some unknown agent in the blood arrests the function of the bulbar and spinal motor centres. Until the nature of the disease is discovered treatment can be only symptomatic.

Prognosis. The prognosis in bulbar paralysis is very serious. The patients die within three years of the onset of the disease, and some cases run a more rapid course. The length of life may be prolonged by care in feeding these patients.

Treatment. The treatment should consist in keeping up the nutrition and general health of the patient by every possible means. Good food, massage, life in the open air, stimulating water treatment by warm packs or hot baths, followed by cool showers or douches, and all forms of tonics should be advised. Strychnine, arsenic, and phosphorus in the form of hypophosphites and the glycero-phosphates should be employed freely and kept up, each drug being used a short time, and those selected and continued which seem to agree best. In the early stage the patient should be carefully instructed in the passage of the stomach-tube, and this must be insisted upon, as later in the disease, when difficulty in swallowing begins, the chief danger can be avoided by its use. When solids can no longer be swallowed semisolid food should be given and supplemented by eggs and milk and broths given through the tube. When nothing can be voluntarily swallowed all food has to be given in this way. I have known a patient to be kept alive over a year by this method of feeding. The thirst can be reduced by large enemata of salt solution and by following each feeding by giving water through the tube. In the last stages of the disease hypodermic medication, with morphine to allay the mental distress, with atropia to prevent the undue flow of saliva, and with strychnine to support the vital centres may be employed.

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[1] See Brain, 1900. Oppenheim, Die myasthenische Paralysie, Berlin, 1901.

CHAPTER XXXIV.

THE CRANIAL NERVES AND THEIR DISEASES.

THE number, name, and function of the cranial nerves are as follows:

First. Olfactory, sense of smell.

Second. Optic, sense of sight.

Third. Oculomotor, motion of the eyeball up, down, and in.

Fourth. Patheticus, motion of the eyeball in and up.

Fifth. Trigeminal, sensory nerve of the face; probably nerve of taste; and motor nerve of the muscles of mastication.

Sixth. Abducens, motion of the eyeball outward.

Seventh. Facial, motor nerve of the face.

Eighth. Acoustic; cochlear division, nerve of hearing; vestibular division, nerve of equilibrium.

Ninth. Glossopharyngeal, nerve of the throat, sensory and motor; possibly of taste.

Tenth. Pneumogastric or vagus nerve, larynx, and vital organs.

Eleventh. Spinal accessory, nerve of the larynx and neck.

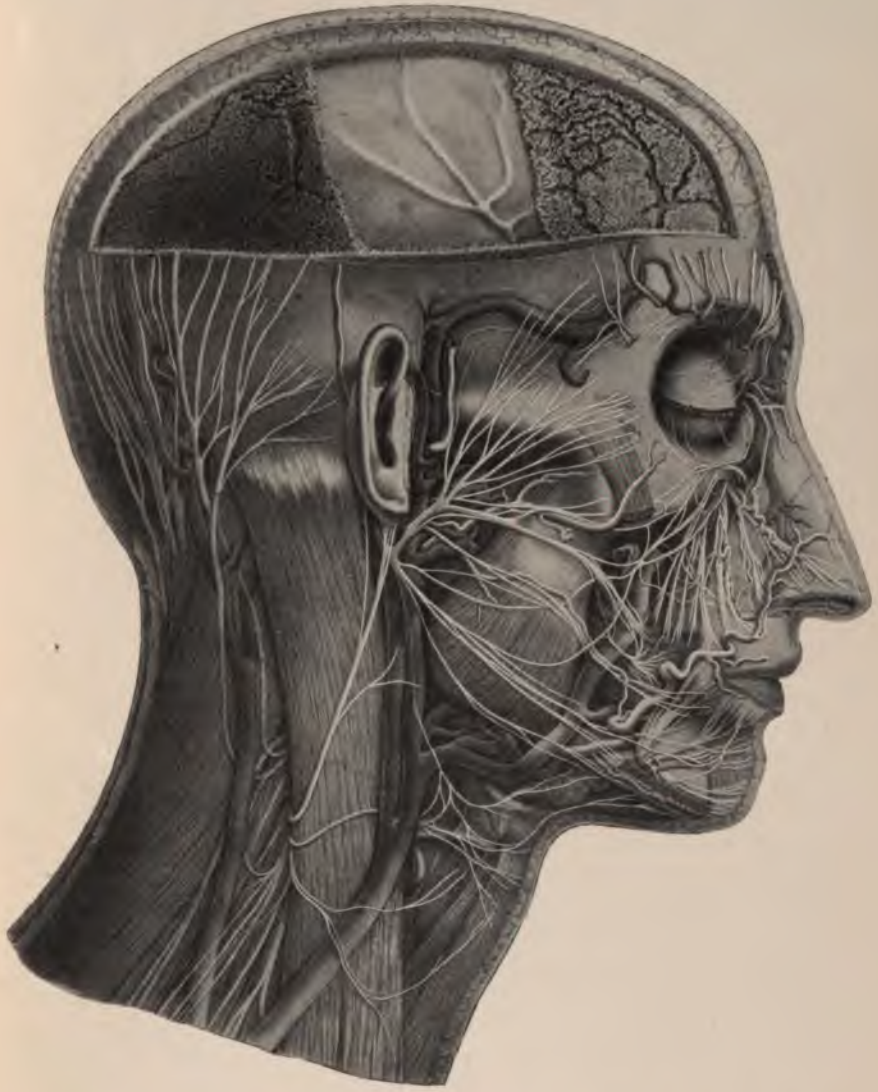
Twelfth. Hypoglossal, nerve of the tongue.

Each of these nerves has a peripheral course from the surface of the head, from some organ of sense, or from some group of muscles to its primary nerve centre lying upon the base of the brain. Plate XXIII. shows the course and the distribution of the superficial nerves of the head and face. The deeper origin of the cranial nerves is shown in Figs. 178 to 182, 198, 199, 241, and 245.

The primary centres or cranial nerve nuclei are closely joined to one another by association fibres, and their interaction is necessary in many reflex acts, such as the sniffing of an odor or a sneeze, the turning of the eyes and head toward an object seen, the winking caused by dust in the eye; the motions of the face in smiling, frowning, or crying; the pose of the head in listening, or quick turning toward a sound, or a start with winking at a noise; the involuntary swallowing of saliva or of tasting food, and the acts of respiration and coughing.

Each cranial nerve is also connected with the higher centres of the brain. Some are joined directly to the brain cortex. The majority have an indirect connection, the tract from the primary centre to the cortex being interrupted in the basal ganglia. This interruption is for the purpose of providing for the numerous unconscious and invol-

PLATE XXIII.

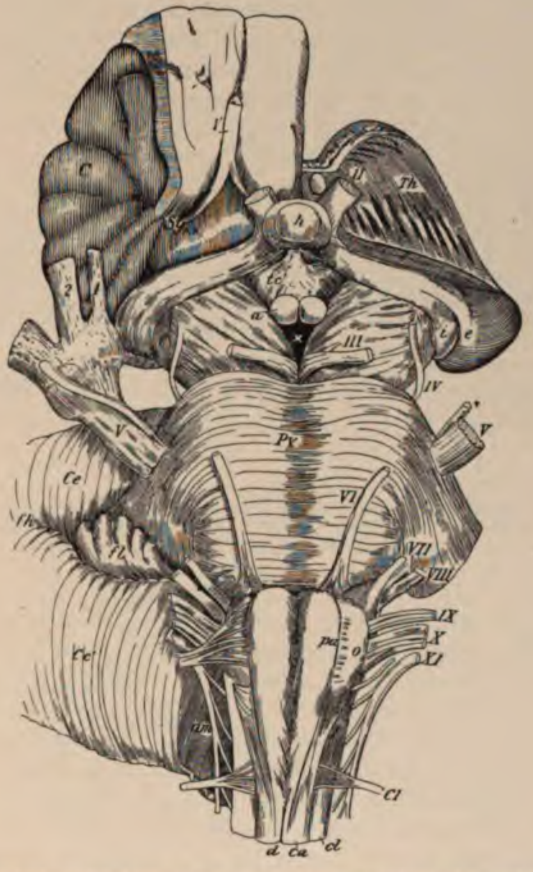


Showing the distribution of the fifth, seventh, and eleventh cranial nerves and the cervical and brachial plexuses; also the area of the middle meningeal artery in the inner table of the skull, injury to which is sometimes the cause of hemiplegia; also the course of the bloodvessels in the neck and face. (Arnold's Atlas.)

untary automatic acts of high complexity which involve the co-ordinated action of numerous widely separated centres.

In studying the cranial nerves it is evident, then, that a sharp distinction must be made between diseases in the peripheral part of the nerve, diseases in the primary centres of the nerve, diseases in the intracerebral tracts of the nerve, and diseases of the secondary or cortical centres of the nerve. In this section the diseases of the peripheral part and of the primary centres of the cranial nerves are considered. The diseases of the intracerebral tracts and cortical centres have been treated under brain diseases, pages 422 to 475.

FIG. 245.

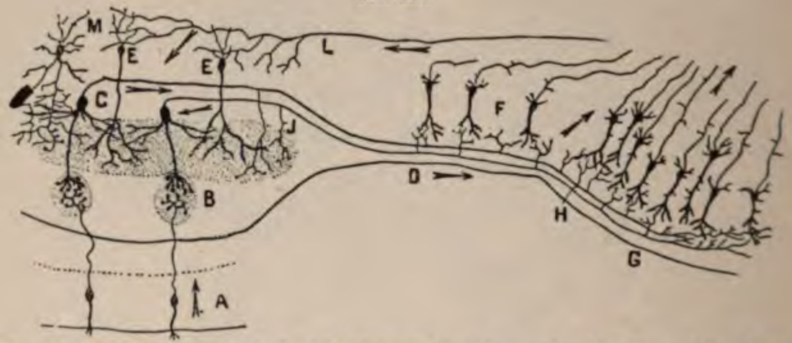


The base of the brain and the crura, pons, and medulla, showing the superficial origin of the cranial nerves. *I* to *XII*. The cranial nerves. *Th*. Optic thalamus. *h*. Pituitary body. *tc*. Tuber cinereum. *a*. Corpora albicantia. *P*. Pes pedunculi. *i*. Interior and *e* exterior geniculate body. *PV*. Pons Varolii. *pa*. Anterior pyramid of medulla. *o*. Olive. *d*. Decussation of anterior pyramids. *ca*. Anterior column of spinal cord. *cl*. Lateral column of spinal cord. *Ce*. Cerebellum. *fl*. Flocculus of cerebellum. (Allen Thompson.)

THE FIRST NERVE: THE OLFACTORY NERVE.

The structure and course of the olfactory nerve to the olfactory bulb and from the bulb to the brain is demonstrated in Figs. 246 and 191. The primary neurones and the finer filaments of the nerve endings are situated in the Schneiderian membrane on the upper and middle turbinated bones and on the septum of the nose, all of which parts are pigmented. It is in the olfactory bulb that the primary sensory neurones terminate in a fine brush about the glomeruli of the nerve. (Fig. 246.) The course from the olfactory bulb to the brain must be regarded as an intracerebral tract. The mitral cells in the bulb are the secondary neurones which receive impulses from their dendrites about the glomeruli and send impulses along their axones in the olfactory tract to the cortex. Fig. 191 shows the course of this tract to its various terminal stations in the optic thalamus and in the cortex of the uncinate gyrus. The thalamic centre is joined to the cortex by the fornix and fascia dendata. It is from the study of comparative anatomy that these facts are established. In man the olfactory apparatus has been reduced to an almost rudimentary state.

FIG. 246.



The olfactory bulb and tract. A. Schneiderian membrane in nose in which lies peripheral olfactory neurone. B. Glomerulus of olfactory bulb. C. Mitral cells with dendrites in B and axones in D olfactory lobe. E. Granule cells. F. Cells in olfactory lobe. H, G. Fibres of olfactory tract. (Ramon y Cajal.)

Any disease that affects the terminal filaments of the nerve in the mucous membrane of the nose will suspend the sense of smell. This symptom is termed *anosmia*. Acute or chronic catarrhal affections of the nasal organs, arrest of the nasal secretion, which occurs when the fifth nerve is diseased; tumors of the nose, or polypi; caries of the ethmoid bones, or ethmoiditis, or diseases which interrupt the transmission of impulses to the olfactory bulb will cause anosmia. Meningitis upon the base of the brain, fractures of the base of the skull, and tumors of the bone or of the brain in the anterior fossa are capable of tearing or destroying the fine filaments of the olfactory nerve or of separating them from their connection with the bulb.

Any disease which destroys or compresses the olfactory bulb or tract or its terminal station in the cortex will have the same effect. An arrest of activity of this centre occurs in hysteria.

The sense of smell has much to do with our appreciation of taste, and when it is lost it is impossible for the patient to perceive very delicate differences of taste in various substances. This may be the symptom first noticed by the patient. Loss of the sense of smell is a symptom which is not often perceived spontaneously as long as the disease is unilateral; hence it has to be sought for by testing the patient with various fragrant substances. The tests are best made with various perfumed powders or essential oils, for it is to be remembered that ammonia, alcohol, and vinegar, which are commonly used for this purpose, produce irritation of the fifth nerve as well as of the first, and should not be used as tests. The symptom is of interest chiefly as an indication of disease in the anterior fossa of the skull, such as tumor or abscess of the brain. A primary isolated neuritis of the first nerve has not yet been recorded. It occurs only as a result of nasal disease. Atrophy of the olfactory nerve has been said to occur as a complication of tabes. It must be the terminal lesion in any destruction of the nerve fibres. Irritation of the olfactory organ or of its cortical centres may cause hallucinations of smell (parosmia). This is seen in epilepsy and in insanity. (See page 460.)

THE SECOND NERVE: THE OPTIC NERVE.

Anatomy. For the finer anatomy and physiology of the optic apparatus and retina the reader is referred to the text-books on ophthalmology.

The anatomy of the optic nerve and its connections with the brain are shown in Fig. 247.

Many primary optic neurones are situated in the retina, their dendrites being connected with the rods and cones, and these send their axones to the primary optic centres in the thalamus, corpora geniculata, and corpora quadrigemina. It will be noticed that some cell bodies in these basal ganglia send their axones outward to the retina, where they terminate in the rods. Thus the optic nerve contains fibres which pass in both directions. The optic nerves pass backward from the eye to the optic chiasm, where a partial decussation of the fibres occurs, the majority crossing over to the other side to enter the opposite optic tract and the minority turning directly into the optic tract of the same side. The crossing fibres convey impulses from the inner or nasal half of the retina, and hence their injury causes blindness in the outer or temporal portion of the visual field, which is much the larger portion. Fibres from the macula lutea, which convey central visual impulses, pass from each eye to both optic tracts; hence central or direct vision is never affected by a lesion in the optic tract. (See Fig. 248.) The optic tracts, containing fibres from both eyes, curve about the crura cerebri and terminate in the primary optic centres, as shown in the diagram. A lesion of one optic nerve causes blindness of one eye. A lesion of

FIG. 247.

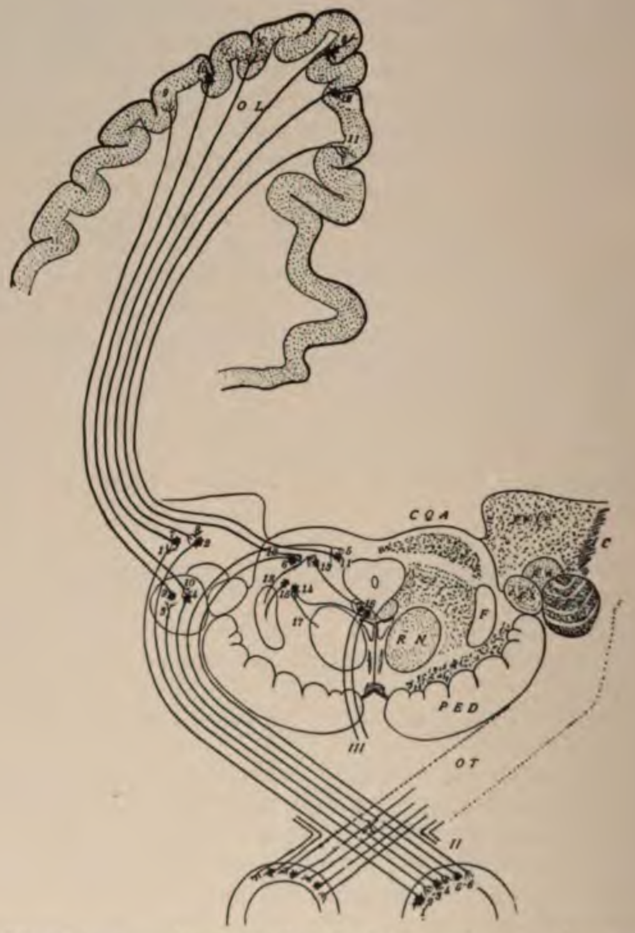
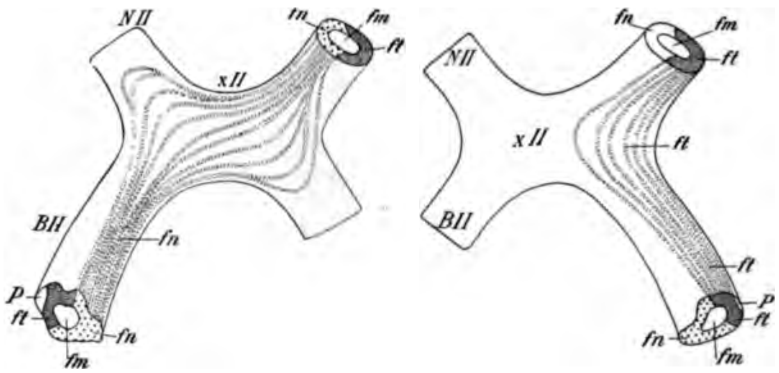


Diagram of the optic nerves and tracts. On the right of the figure the masses of gray matter in the basal ganglia are shown. *Pulv.* Pulvinar of the optic thalamus. *Pn.* Posterior nucleus of the optic thalamus lying between the corpus geniculatum externum *cge*, and the corpus geniculatum internum *cgi*. *IC.* Internal capsule. *F.* Fillet. *RN.* Red nucleus of the tegmentum. *PED.* Peduncle of the cerebrum. *Sn.* Substantia nigra. *OT* Optic tract. *X.* Optic chiasm. *II.* Optic nerve. 1, 2. Fibres from the retina to the pulvinar of the optic thalamus. 1. Centripetal. 2. Centrifugal. 7, 8. Fibres between the optic thalamus and the occipital cortex. 3, 4. Fibres between the retina and the corpus geniculatum externum. 9, 10. Corresponding fibres to the occipital cortex. 5, 6. Fibres between the retina and the corpus quadrigeminum anterior. 11, 12. Corresponding fibres to the occipital cortex—for the sake of distinctness the decussating optic fibres only are traced. 13. Cell of the superficial gray matter of the *CQA* passing to the nucleus of the third nerve (16). 14. Cell of the deep gray of *CQA* passing to the third nerve nucleus. 15. Cell of the deep gray of *CQA* sending fibre into the fillet. 17. Fibre from the red nucleus, terminating about 14. 18. Fibre from the fillet, terminating about 13. *OL.* Occipital lobe of the brain with its cortex, containing both cells and terminal brushes of the visual tract. (Starr, Atlas of Nerve Cells.)

the optic chiasm causes partial or total blindness, according to the number and position of the optic fibres destroyed. If the lesion involves one lateral part only of the chiasm (Fig. 248, *ft*), the fibres from the temporal half of the retina, are involved, and a unilateral nasal hemianopsia is caused, that is, a blindness in the inner portion of the visual field of one eye. If the lesion involves the anterior or posterior part only of the chiasm (Fig. 183, *T* or *H*), the fibres which come from the nasal halves of the retinæ are involved, and a bilateral temporal hemianopsia is caused, that is, a blindness in the outer portion of the visual field of both eyes. This is termed heteronymous hemianopsia. If the lesion involves the optic tract after the decussation a blindness in one-half of both eyes is caused, and the patient is deprived of seeing anything to one side of the middle line. This is termed homonymous hemianopsia.

Fig. 248.



The relation in the optic nerves, optic chiasm, and optic tracts of the fibres from the nasal half (*fn*), temporal half (*ft*), and central spot (*fm*) of the retina. *NII.* Optic nerve. *BII.* Optic tract. *xII.* Optic decussation. The left figure shows the fibres which cross; the right figure shows those which do not cross. *P.* Pupillary fibres. (Dejerine)

In none of these forms of hemianopsia is central vision impaired. It is only affected in lesions of the nerve itself. The acuteness of vision is best tested by Snellen's test types, type No. xx as seen at twenty feet being taken as indicating normal vision $\frac{xx}{20}$.

The extent of the visual field is to be determined for each eye separately. This is to be done by having the patient fix each eye in turn on that of the examiner, the other eye being covered, and then notice whether he can see the hand of the examiner or a small white object held by him as it is passed outward from the centre of vision on all sides. The retina is less sensitive in its peripheral parts, and the less the illumination of the test object the less distinctly is it seen. The luminosity of blue, red and green are less than white in the order named; hence the visual field is greatest for white, next for blue, next for red, and least for green.

Contraction of the visual field for white or colors as well as defect in the visual field is an important symptom.

The optic nerve contains some fibres which pass to the motor mechanism controlling the action of the iris. (Fig. 248, *P.*) Hence impressions of light cause a narrowing of the pupil. If one optic nerve only is destroyed the pupil reflex is lost in that eye when it is exposed to light. But as this reflex is bilateral, light in the unaffected eye will cause a contraction of the pupil in the blind eye. The fibres which subserve this pupil reflex decussate at the chiasm. Hence if one optic tract be injured, its function suspended, we have not only a bilateral hemianopsia, but also an interference with the pupil reflex. This is difficult to determine, for, as already stated, the reflex is a bilateral one, and light thrown on the unaffected parts of one of the retinæ is capable of causing a prompt narrowing of both pupils. If, however, the patient be examined in a dark room, the eyes be only faintly illuminated, so that the pupils are fairly dilated, and then a ray of light be thrown by a mirror from one side carefully upon the blind side only of the retina, no pupil reflex will ensue. This indicates infallibly a lesion of the optic tract, or basal ganglia, as it does not occur in cases where the hemianopsia is due to lesion elsewhere in the visual tract, as, for example, in the occipital lobe. It is termed Wernicke's test for a hemiopic pupillary reflex.

In lesions of the optic tract the hemianopsia affects color sense as well as white and object vision. It is only in affections of the visual centres in the cortex of the cerebral hemisphere that hemianopsia for color, with preservation of sight for white and objects, has been observed. This is called hemichromatopsia. (See page 449.)

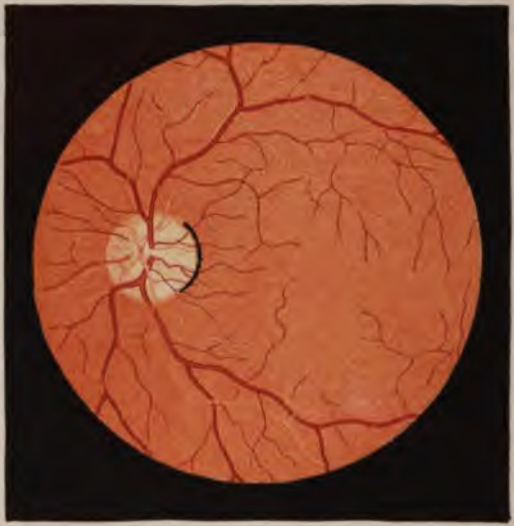
Diseases of the Optic Nerve.

Hyperæmia and **anæmia** of the optic nerve accompany corresponding conditions of the brain, but unless very extreme, are not easily recognized, even by experts, as the normal tint of the retina and the normal calibre of the bloodvessels differ in different individuals within very wide limits, like the color of the hair and of the skin.

Gowers states that a state of hyperæmia may be admitted when the disk is dull red or brick color and when the edge of the disk cannot be distinguished from the rest of the retina. Such a condition is usually associated with a hypersensitiveness to light and pain on using the eyes. The causes of hyperæmia are overaction or strain of the eyes due to refractive errors, or the use of the eyes in intense glare and heat, or inflammation of the conjunctiva, iris, or choroid, or some toxæmia, or some form of cerebral or spinal disease. The condition has been observed in many different types of insanity, but no form of insanity is particularly characterized by this symptom. It is a matter of dispute whether the chronic forms of spinal disease which lead to atrophy produce a preliminary hyperæmia or anæmia. I have never observed any indication of either condition in cases which I have observed.

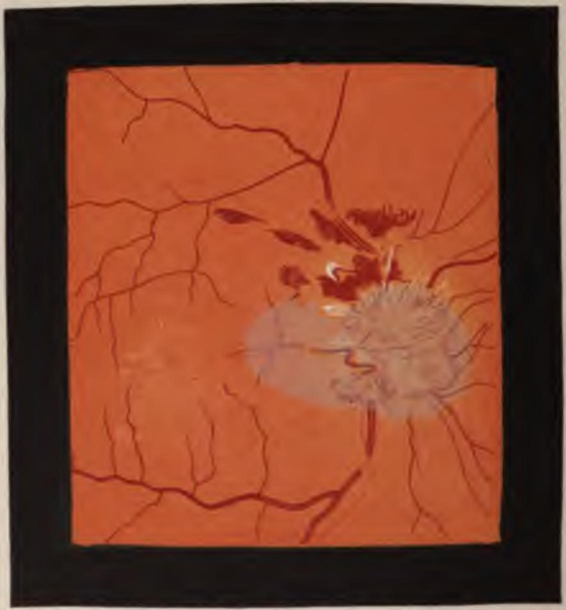
PLATE XXIV.

FIG. 1.



Normal Eye-ground (average tint). (Norris & Oliver.)

FIG. 2.



Ophthalmoscopic Appearances in Early Stage of Optic Neuritis. (Norris & Oliver.)

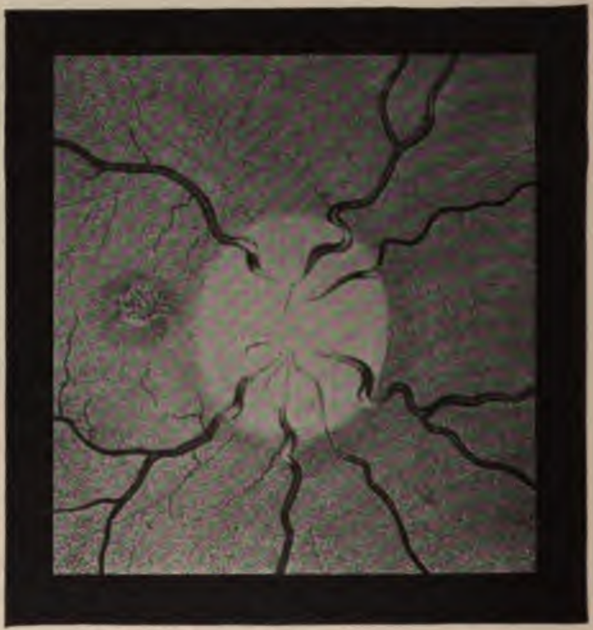
The pallor of anæmia is almost as great as that of atrophy, and in the early stage of atrophy they cannot be distinguished. The causes of anæmia of the disk are those of general anæmia. A sudden anæmia due to embolism of the central artery of the retina always causes sudden blindness in one eye, and the contrast between the appearance of the two eyes serves to establish the existence of unilateral anæmia. The arteries in one retina are seen to be small and empty. A moderate degree of anæmia may be caused by disease of the bloodvessels and may lead to temporary blindness. This was the case in a woman sixty years old, who had aortic obstruction and extensive general endarteritis not specific in origin. She had suffered from unilateral headaches for some years. On November 1st she took a coal-tar remedy for headache, which probably depressed the heart, and within a few hours she noticed a great dimness of vision in the right eye. This increased rapidly; in two days she was nearly blind, and on December 1st the right eye was totally blind. Three days later vision began to return in this eye, and by January 1st was fairly good. But during the latter part of November the left eye began to fail, and by the middle of December, while the right eye was recovering, the left eye was totally blind. In February the left eye also improved, and she finally recovered vision in both eyes. During all this time the disks were pale, the arteries small but not obliterated, and the veins were distended. She could see better when lying flat on her back than when standing. The absence of visible neuritis, the marked arterial anæmia, and the existence of general endarteritis with aortic obstruction, and the recovery, prove the symptoms to have been due to circulatory conditions. The treatment, which consisted of full doses of iodide of potassium, 90 grains daily, with strychnine and digitalis, was attended not only by relief of the blindness, but also of the headaches, which were probably due to the same cause.

Thrombosis of the retinal artery may cause local anæmia of the retina, and may be followed by œdema and hemorrhagic effusion, a condition exactly like that which occurs in the brain. This was the condition found in the following case: A boy, aged thirteen years, had had a continued fever for six weeks with temperature between 99° and 102° F., supposed to be enteric, but probably malarial, as the blood examination showed the presence of two generations of tertian malarial parasites. He then developed dimness of vision in the left eye and then in the right. His optic disks were swollen three diopters, with flame-like appearance about the disk. In the left eye a wedge-shaped area extended up and in from the nasal side of the disk, and was colorless; the retinal artery on this side was small; the veins were normal, numerous dots about the macula indicating plastic exudation. This condition slowly improved. Later a marked picture of thrombosis of the retinal veins developed in the right eye, with a narrowing of the arteries and much plastic exudation about the disk, and a few retinal hemorrhages. This too gradually subsided, and after four months the optic nerves presented a fairly normal appearance and his vision had returned. The origin was presumably malarial infection, as nephritis was not present, and there were never any signs of cerebral disease.

I have seen thrombosis of retinal arteries associated with symptoms of general cerebral arterial disease which subsequently caused death. It is to be regarded, therefore, as a condition of prognostic importance as indicating a widespread arterial disease. The treatment must always be directed to a removal of the cause.

Œdema of the Optic Nerve. "Choked Disk." The optic nerve at its exit from the eyeball receives a double sheath, one layer of which is continuous with the dura mater and the other with the pia mater. When fluid accumulates under pressure within the cranium it therefore finds its way into the space between these sheaths, distending the dural sheath and compressing the nerve at its exit from the eyeball. Such compression is felt earlier by the lymphatics and veins than by the harder nerve fibres, and the consequent result is a dropsical effusion into the intraocular portion of the nerve from transudation of fluid from the obstructed vessels. This causes a swelling of the optic disk visible to the ophthalmoscope and causing an appearance shown in Fig. 249. Such a swelling may exist for some time without any impairment of vision, hence the importance of ophthalmoscopic examinations in cases without ocular symptoms.

FIG. 249.



Swollen disk in a case of chronic meningitis. (Liebreich.)

The experiments of Merz[1] have demonstrated that this swelling of the nerve may come and go rapidly and may vary greatly in degree, according to the varying amount of intracranial pressure. The œdema is always attended by venous congestion, but no inflammatory changes may occur, and it is therefore erroneous to identify choked disk with optic neuritis. It is not infrequently accompanied by hemorrhages in the retina. Choked disk will occur under any condition which obstructs the circulation in the eye or increases the intracranial pressure. It is very common in tumors and abscesses of the brain, occasionally occurs temporarily after large hemorrhages, and not infrequently accompanies Bright's disease or those affections in which general œdema is a symptom.

The only positive means of diagnosis is by ophthalmoscopic examination. In many cases when the choked disk has lasted for some time vision is impaired and the visual field is diminished in extent. In a few cases it produces no symptoms. In some cases it causes a rapidly advancing blindness. But in any case this blindness may pass away as the œdema of the nerve subsides, provided a true neuritis or atrophy does not ensue. Choked disk is usually bilateral, but may commence in one eye some time before it appears in the other. In some cases it goes on to neuritis and to atrophy. Cases have been recorded of choked disk without other symptoms and without apparent cause. In two such cases, reported by Brudenel Carter,[2] unsuspected tumor of the brain and latent abscess of the sphenopalatine fossa were eventually proven to be the cause.

Optic Neuritis. Etiology. Optic neuritis occurs occasionally without apparent cause, and must be regarded as an idiopathic affection. It has been known to be hereditary, and develops about the age of twenty and goes on to blindness. In the majority of cases, however, it is secondary to other conditions. It may be due to general poisons in the blood, such as produce multiple neuritis, namely, alcohol, lead, arsenic, mercury, and tobacco. It is the pathological state which is present in so-called toxic amblyopia and in the affection described by ophthalmologists as retrobulbar neuritis; where the fibres passing from the macula lutea are especially involved. It may occur as a sequel of any of the infectious diseases, especially malaria. It also occurs occasionally as a symptom of severe chronic anæmia, of pernicious anæmia, leucocythæmia, and diabetes. It may be syphilitic in origin. It is a frequent symptom of nephritis. It is supposed to be caused by rheumatism, by gout, or by taking cold. Optic neuritis may be secondary to a meningitis, in which case the inflammation extends directly along the sheath of the nerve. Its most common cause, however, is from pressure within the skull exerted either directly by tumor or abscess of the brain, or syphilitic exudations upon the base of the brain, or by the pressure of fluid in the ventricles. Much discussion has been devoted to the cause of optic neuritis in intracranial disease, and no definite conclusion can yet be reached as to the actual causation of this affection. It may be due to the increase of pressure within the head, producing œdema of the nerve trunk and a secondary neuritis, as Parinaud affirms. It may be due to a direct infection of the nerve by products of disease within the brain, as Leber and Deutschmann have long maintained. In all these cases optic neuritis is usually bilateral, though in tumors of the brain, upon the base, with direct pressure upon the nerve, it is possible for the nerve on the side of the tumor to be affected much sooner than the other.

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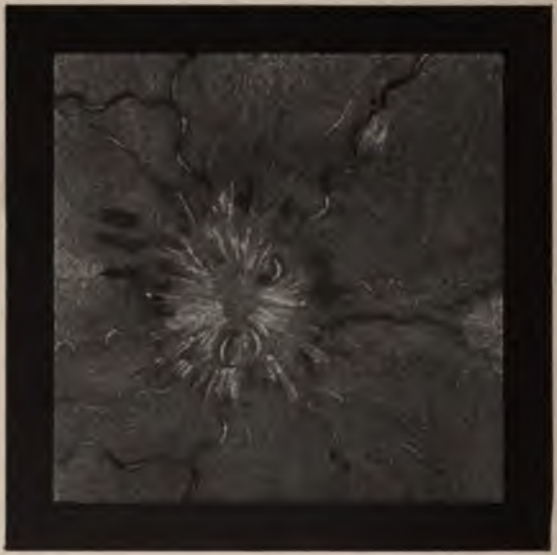
[1] Merz. Arch. of Ophthalmology, July, 1901.

[2] Clifford Allbutt's System of Medicine, vol. vi. p. 842.

Symptoms. The symptoms of optic neuritis are a sudden or gradual failure of sight, objects becoming blurred, as if a fog were between the eye and the object. Some patients complain of sensitiveness to light and of distress in using the eyes. There is also a diminution of the visual field and a lack of distinctness of color vision, blue being lost first. In some cases irregular blind spots develop in the visual field. It is rare for total blindness to occur as the result of optic neuritis within six months of the onset. The progress of the blindness is continuous unless the cause can be removed.

FIG. 250.



Acute optic neuritis. Note disk much swollen; estimated at +7 D. Edge indistinct; vessels obscured at edge; large number of hemorrhages around the disk, patches of œdema in the retina. veins very tortuous. (Posey.)

Diagnosis. A diagnosis can be made only by an ophthalmoscopic examination. The figures on Plate XXIV. show the appearance of a normal retina and of a nerve in the early stage of optic neuritis. (See also Fig. 250.) It will be noticed that the changes are very marked in the bloodvessels of the retina, the arteries being smaller and the veins being distinctly congested. Their point of exit in the disk is often obscured. It will be noticed that the outline of the disk in the early stage is no longer clear and in the later stage is completely obliterated. The peculiar striated appearance about the disk is quite evident in the figures, being more marked in the later stage of the disease, and this striation is partly yellowish and partly red, in contrast to the uniform color of the retina in health. In the later stage hemorrhages in the retina are evident, being indicated by red or yellowish-white plaques. Small whitish or yellow spots are also seen which are due to exudation of inflammatory products. There is an actual exudation within the nerve trunk, so that the optic disk is actually swollen. Its height can be estimated by the ophthalmoscope (1 diopter equals $\frac{2}{3}$ mm.), but the first evidence of this swelling is the disappearance of the clear central space known as the physiological cup. If the condition subsides there is a gradual reduction of this swelling and little by little a return to the normal appearance of the disk. While the diagnosis of the existence of optic neuritis is easily made by the aid of the ophthalmoscope, it is by no means easy to determine in every case to what the condition is due. If to the symptoms of neuritis there are added headache, malaise, attacks of vertigo, disorders of digestion, with nausea and vomiting, and some irregularities of circulation, and the patient's health, strength, and nutrition suffer the question at once arises whether these are the symptoms of tumor of the brain, chronic tubercular meningitis, general arterial disease, nephritis, or merely of general anæmia. I have seen two patients suffering from these symptoms during several months and not a little distressed mentally lest they might be developing a fatal affection. Both were young women suffering from anæmia and also from very irregular painful menstruation due to endometritis. But in both the severity of the nervous symptoms had awakened the fear of brain tumor. Both eventually recovered under general tonic treatment. In such cases the diagnosis can be reached only after a considerable time, the lack of development of local brain symptoms, especially in the cranial nerves, the existence of normal powers of sensation and motion, and of normal reflexes being negative symptoms of great value. On the other hand, the appearance of localizing brain symptoms may confirm the suspicion that the neuritis was the first sign of serious intracerebral disease, especially of tumor or abscess. I have seen this condition as an early symptom of serious arterial disease, which led rapidly to multiple cerebral thrombosis with aphasia and hemiplegia and death. It is well known that a very common cause of optic neuritis is Bright's disease, and it follows that an examination of the urine should be made in every case. It is never to be forgotten that abuse of alcohol and tobacco combined may be the cause of optic neuritis and may also produce other nervous symptoms. I have seen such a condition in young women addicted to champagne and cigarettes, as well as in many young men of nervous constitution who were victims of the same habits. It is evident, therefore, that in any case of optic neuritis great caution should be exercised in giving a prognosis, every etiological factor should be investigated, and the course of the case should be watched with extreme care. The lesion in optic neuritis is similar to that already described as a diffuse neuritis (page 42), there being a congestion of the vessels, an effusion of serum and blood cells, of leucocytes, and nuclei in the endoneurium, as well as a parenchymatous degeneration of the nerve fibres.

Prognosis. The prognosis in the affection will depend entirely upon the possibility of removing the cause. Even when the optic neuritis has been quite extreme recovery is still possible. Thus, after absorption or removal of tumors of the brain I have frequently seen recovery of vision when the blindness had been so extreme as to incapacitate the patient from recognizing even the largest letters upon the test card.

Treatment. The treatment for the affection is the removal of the cause, if possible, and complete rest of the eyes, which should be shaded from the light. The ophthalmologists prescribe strychnine by hypodermic injections or by the mouth in large doses, $\frac{1}{40}$ grain, increased at each injection until in ten days $\frac{1}{4}$ grain is given, and this drug will certainly stimulate the nerve to activity, even when in a state of neuritis. Whether such stimulation is desirable may be open to question, and certainly this treatment has no effect in other forms of neuritis, excepting a temporary effect of stimulation of the functions of the nerve. The use of mercurial inunctions and of full doses of iodide of potassium is recommended both in syphilitic and non-syphilitic cases. The local depletion by wet cups or leeches to the temple is often beneficial in the early stage. In anæmic cases general tonics are of benefit.

Optic Nerve Atrophy. **Etiology.** Optic neuritis in many cases goes on to a gradual atrophy of the optic nerve, which is then spoken of as consecutive, and in such cases the atrophy is the terminal result. Increased pressure within the cranium may produce atrophy without any primary neuritis, the nerve appearing to undergo a degeneration from the start, there being no initial process of congestion, such as occurs in neuritis. A secondary atrophy also occurs after lesions of the nerve in the orbit or after affections of the optic chiasm and tract. Optic atrophy may, however, occur as a primary affection without any preceding intracranial pressure or disease. Some cases are congenital or develop very soon after birth in infants of syphilitic parentage or from actual congenital defective development of the optic nerve. In these cases other nervous symptoms usually indicate an imperfect development of the nervous system, such as imbecility and idiocy, or spastic paralysis. These cases have been described most fully by Sachs.[1] In many cases primary optic atrophy is a condition

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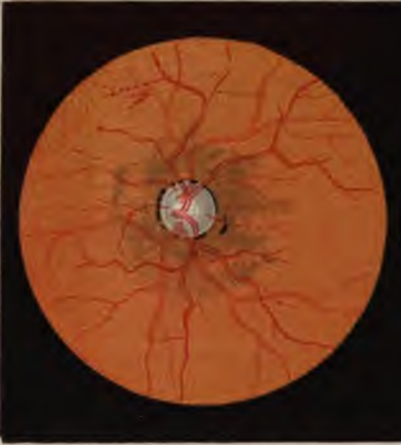
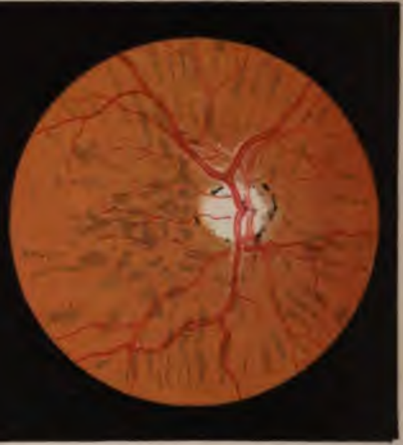
[1] Amaurotic Family Idiocy, Journal of Nervous and Mental Disease, 1887, p. 541, and 1903, p. 1.

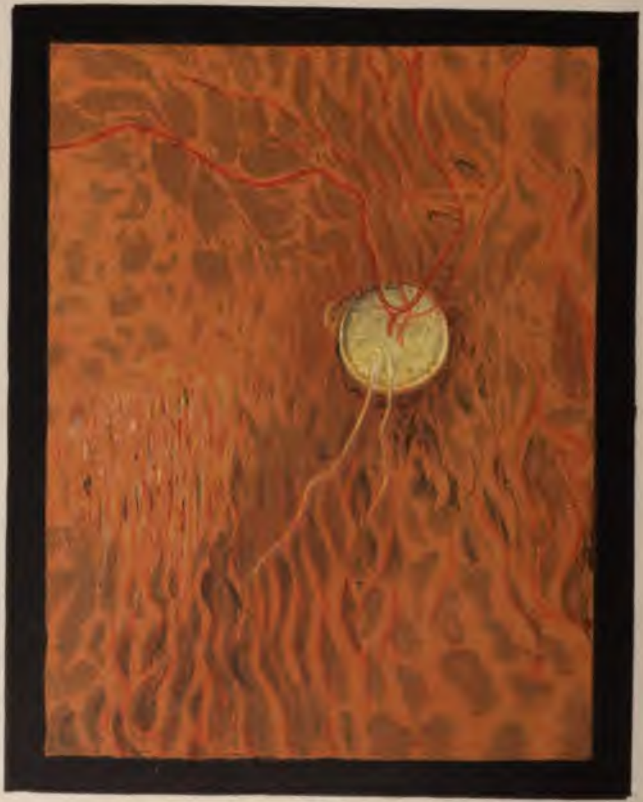
PLATE XXV.



Primary Atrophy of Optic Nerve (Spinal Atrophy). Modified from Haab.



Post-papillitic or Consecutive Atrophy of the Optic Nerve. Modified from Juler.



Embolic Atrophy of the Optic Nerve. (De Schweinitz, in Dercum's Nervous Diseases.)

associated with other diseases, though it can hardly be called secondary to them. This is notably the case in locomotor ataxia, in disseminated or multiple sclerosis, and in paresis. In all of these diseases the occurrence of optic atrophy is an exceedingly serious complication and probably due to the same cause which produces the original affection. It may be the first lesion produced by this cause. Thus in locomotor ataxia 10 per cent. of the cases begin with optic atrophy. And in multiple sclerosis 52 per cent. of the patients develop this complication. Optic atrophy may occur as the result of poisoning by tobacco, alcohol, lead, and quinine. It may occur as the result of the various infectious diseases and of diabetes, and in some cases has been ascribed to cold.

Symptoms. The symptoms of optic atrophy are a gradually increasing blindness, a diminution of the visual field, progressively increasing from without inward, so that finally only central vision is possible, and very often a defective color vision, the fields for various colors being either progressively reduced in extent or altered in their extent. In some cases irregularly situated blind spots, sometimes central, develop—the so-called central scotoma. Pain in the eyes and discomfort after their use or after exposure to strong light are often felt.

The appearances in optic atrophy are demonstrated in Plate XXV., where both gray and white atrophy are shown. The ophthalmoscopic appearances in optic nerve atrophy are not uniform. When the atrophy is secondary to the optic neuritis there are, first, the ordinary changes observed in the neuritis, and then, as the swelling and congestion subside, the disk once more becomes evident, has a clearer and clearer outline, but an unusually white appearance, until finally it presents a perfectly white spot in the retina. The arteries are noticeably diminished in calibre. When the optic atrophy is primary and is not preceded by a neuritis there is a gradual progressive change in the appearance of the normal disk, which becomes more sharply outlined, loses its pink tint, and becomes more and more gray, or even clear white in color, the arteries and veins preserving their normal calibre. After a time the lamina cribrosa at the bottom of the disk may become evident, the appearance presented resembling a fine sieve. The lesion in optic atrophy is a progressive parenchymatous neuritis without any interstitial change.

Diagnosis. The diagnosis of the underlying condition in a case of optic atrophy is of as great importance as in a case of optic neuritis. Toxic amblyopia should be first excluded. Symptoms of organic brain or spinal-cord disease should be looked for. The mental characteristics of the patient should be considered, and changes indicative of beginning paresis should not be overlooked. And the case should be watched for a long period before giving a diagnosis of primary optic atrophy. More than one-half of the cases eventually develop some form of spinal sclerosis.

Prognosis. The prognosis in optic nerve atrophy is much more serious than in optic neuritis, for while the former may go on to recovery, the latter is progressive, and treatment, unless directed to the cause, is not successful.

Treatment. The progress of the case may be delayed by the use of tonics, by everything which improves the general health, and by the hypodermic use of strychnine, from $\frac{1}{40}$ up to $\frac{1}{4}$ grain daily, the dose being increased slowly, or of the chloride of gold, or nitrate of silver, or of nitroglycerin. Recently hypodermic injections of normal salt solution beneath the conjunctiva have been recommended. They have not been useful in my cases.

THE THIRD, FOURTH, AND SIXTH NERVES: THE MOTOR NERVES OF THE EYEBALL. OPHTHALMOPLEGIA.

The eyeball and eyelid are moved by numerous muscles supplied by these three nerves, as follows:

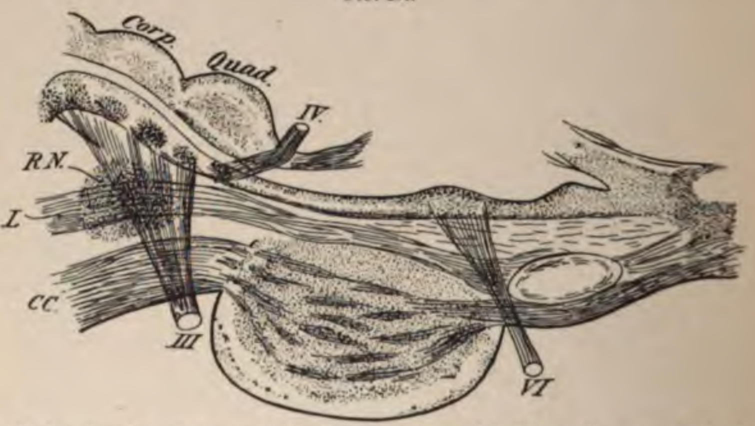
The third nerve supplies the ciliary muscle, sphincter iridis, levator palpebræ, superior rectus, internal rectus, inferior rectus, inferior oblique.

The fourth nerve supplies the superior oblique.

The sixth nerve supplies the external rectus.

The branches of the cavernous plexus of sympathetic nerves supplies the dilator pupillæ. These nerves arise from groups of cells lying upon the floor of the aqueduct of Sylvius and of the fourth ventricle of the brain. (Fig. 251.) The groups are closely joined to each other by association fibres, the longest of which, between the third and sixth nerve nuclei, pass in the posterior longitudinal bundle.

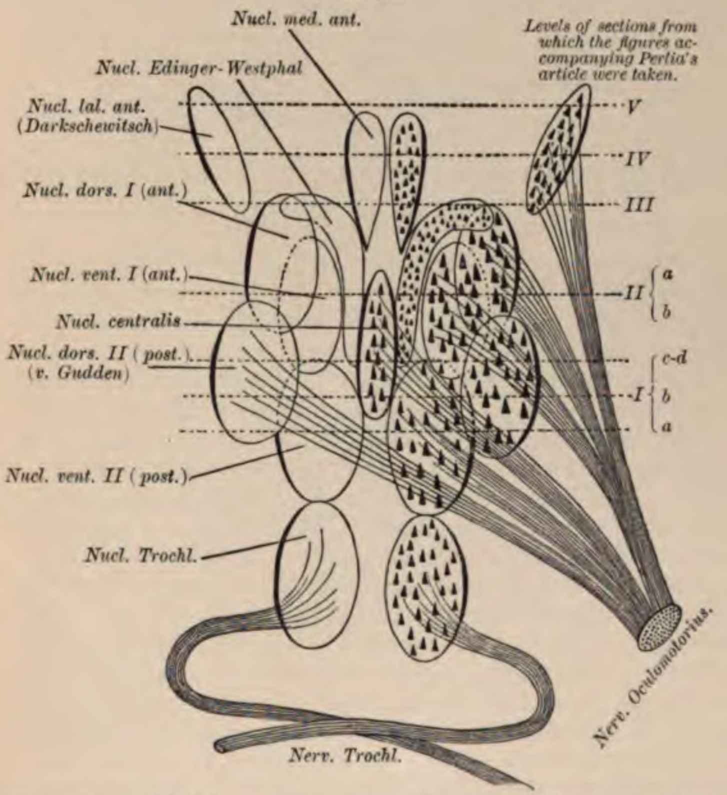
FIG. 251.



Sagittal section through the cerebral axis, to show the nuclei of the ocular nerves in the floor of the aqueduct of Sylvius and the fourth ventricle, and the course of the nerves to their exit. The various groups of cells from which the third nerve arises are seen. *R N.* Red nucleus of tegmentum. *L.* Lemniscus (sensory tract). *C C.* Motor tract in the crus cerebri seen to traverse the pons and enter the anterior pyramid of the medulla.

The location of the nuclei of the oculomotor nerves and the function of each group of cells has been the subject of much discussion, and unanimity of view has not yet been reached, though the subject has been approached from the side of comparative anatomy (Spitzka, Edinger), embryology (von Gudden, Edinger), physiological experimentation (Hensen and Völchers, Bernheim), and pathology (Westphal, Kahler, Pick, and Starr), as well as from the histological standpoint (Kolliker, Perlia, Bruce).

FIG. 252.



Scheme of the nuclei of the oculomotor nerve. (After Perlia, Arch. f. Ophth., Leipzig, Bd. xxv., Abth. iv., S. 297.)

The third nerve nucleus consists of a long column of cells which begin in the gray matter of the wall of the third ventricle near the posterior commissure on the median surface of the optic thalamus, and extends for almost a centimetre in length under the floor of the aqueduct of Sylvius downward to the level of the corpora quadrigemina posterior. Just posterior to this column lies the group of cells giving origin to the fourth nerve, and about 3 cm. below in the pons is the sixth nerve nucleus. The column of cells making up the third nerve nucleus can be divided into a number of groups. These groups, their connections, and their special functions are shown in Figs. 251 and 252. That each group controls a single muscle is proven by the fact that small lesions limited to different parts of the column of cells and affecting certain groups or their nerve roots only produce different forms of paralysis of the eyeball. A study of the combination of paralyses observed in various cases enabled the writer (in 1888[1]) to offer the following scheme of the order in which the muscles are represented in these groups:

RIGHT SIDE.

Median line.	Ciliary muscle.	Sphincter iridis.
	Rectus internus.	Levator palpebræ.
		Rectus superior.
	Rectus inferior.	Obliquus inferior.
		Obliquus superior.
	Rectus externus.	

Subsequent observations have lent support to this localization.

Third Nerve Paralysis.

The third nerve, after arising from its groups of cells upon the floor of the aqueduct of Sylvius, sends its various roots through the tegmentum of the crus cerebri, which traverse on their way the red nucleus of the tegmentum and make their exit in a large bundle on the inner side of the crus in close apposition to the third nerve of the opposite side. Lesions of the groups of cells of origin of the third nerve may produce a partial paralysis of the ocular muscles. So, too, may lesions of the roots of the third nerve in their passage through the crus. But lesions of the nerve after its exit cause paralysis of all the muscles which it supplies. The third nerve passes from its superficial origin in the crus forward to the sphenoidal fissure, and thus into the orbit, where it divides up in the various small branches going to the muscles already named and to the ciliary ganglion.

Etiology. Lesions upon the base of the brain lying near the crus or between it and the exit of the nerve into the orbit are particularly liable to produce paralysis of the third nerve. Meningitis, either simple or tubercular, abscess of the brain, and hemorrhages on the base often involve the nerve. In meningitis in infants it is often the nerve first affected. Syphilitic exudations upon the base of the brain and a syphilitic neuritis with infiltration of the sheath of the nerve are the most common causes of third nerve palsy. Tumors of the brain lying in this locality or lying in such position as to displace the brain axis, and thus cause traction upon the nerve, may also produce third nerve paralysis. In both these conditions other symptoms of the disease accompany the ocular palsy and give a clue to the diagnosis. The oculomotor nerve is rarely injured within the orbit, inasmuch as it is well protected, but occasionally in fractures of the base the nerve will be torn, and severe falls and blows upon the head have been known to cause the bruising of the nerve against a sharp edge of the sphenoidal fissure. Thus in a patient under my own observation a fall from a carriage upon the side of the head produced a sudden paralysis of the third nerve which remained about six months and then gradually recovered. Tumors within the orbit and anything producing extreme exophthalmus may by pressure or stretching involve one or more branches of the oculomotor nerve, and thus cause a partial paralysis. Thus in a patient with exophthalmic goitre and very extreme exophthalmus I have seen an inability to turn the eye inward and upward which subsided when the exophthalmus disappeared. Oculomotor palsy may occur as a complication of diphtheria and of typhoid fever, and, in fact, it has been recorded in many different forms of multiple neuritis; under these circumstances it is usually bilateral. German authorities affirm that a temporary oculomotor palsy may be produced by poisoning with sausage, fish, or meat that is diseased. Lastly, it is supposed that the effect of cold may produce a sudden oculomotor palsy of temporary duration, but this is certainly very rare.

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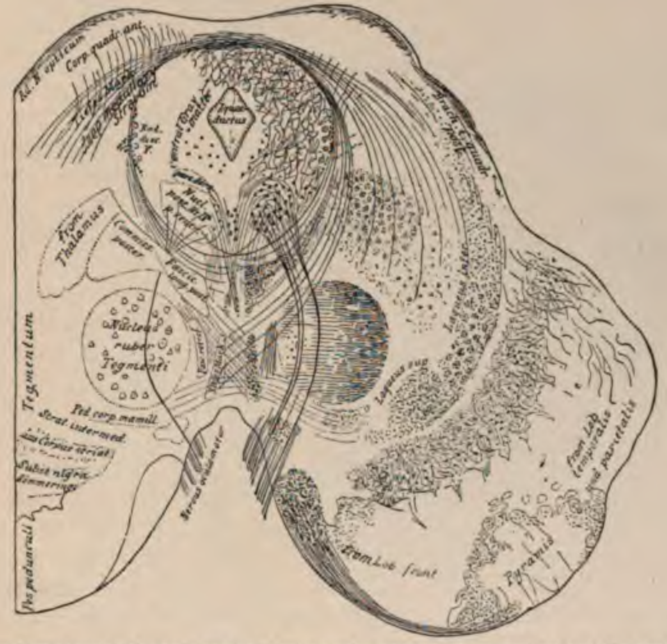
[1] Jour. of Nerv. and Ment. Dis., vol. xv. p. 301.

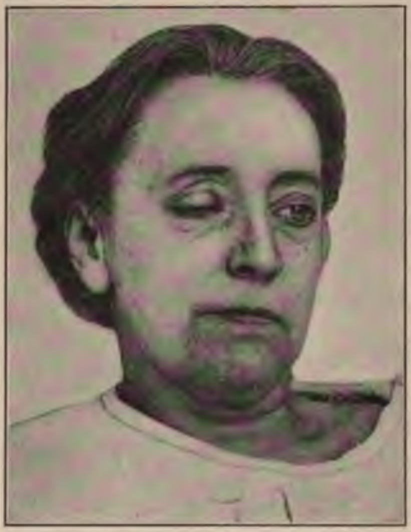
FIG. 253.



A section through the crus cerebri, showing the origin, course, and exit of the oculomotor nerves. (Edinger.)

Symptoms. The symptoms of third nerve paralysis are ptosis or falling of the upper eyelid, dilatation of the pupil, and loss of reflex action both to light and to accommodation and a turning outward and slightly downward of the eyeball owing to the unopposed contraction of the external rectus and superior oblique muscles. The strabismus is attended by double vision and often by vertigo and uncertainty of gait. There is an inability to turn the eyeball upward, downward, or inward, owing to the paralysis of the superior, inferior, and internal recti muscles. The paralysis of the inferior oblique muscle is also present, but does not produce a visible deflection of the eye when the superior oblique is capable of acting. Ptosis is complete, so that the patient is unable to open the eye sufficiently to see, although he usually makes efforts in this direction by elevating the eyebrow to the utmost limit; hence in such patients wrinkling of the forehead is more pronounced on the side of the paralysis. Fig. 254 shows this condition. In cases where the cause can be removed by treatment the prognosis is good, and even in cases of injury and disease of the nerve from cold, recovery, as a rule, ensues. The nerve is inaccessible to any form of electrical treatment, therefore it is impossible to treat the paralysis.

FIG. 254.



Ptosis and external strabismus due to paralysis of the right third nerve. (Icon. de la Salpêtrière.)

The Pupil and Its Nervous Mechanism. The action of the pupil to light and to accommodation is a reflex act in which the optic, oculomotor, and sympathetic nerves take part.

The contraction of the pupil to light is produced by impulses passing from the retina to the corpora quadrigemina anterior; thence to the anterior group of cells in the third nerve nucleus, thence in the third nerve to the ciliary ganglion, from which the short ciliary nerves go to the iris, which contracts by closure of its sphincter.

Any lesion which breaks this reflex arc will arrest the contraction of the pupil to light. The dilatation of the pupil in darkness or in distant vision is produced by impulses passing to the dilator pupillæ through the sympathetic nerves from the cilio-spinal centre in the eighth cervical and first dorsal segments of the spinal cord. These spinal centres are connected with the corpora quadrigemina by nerve fibres which pass in the tegmentum of the pons and medulla and in the antero-lateral columns of the upper cord. The sympathetic nerve fibres leave the spinal cord in the first dorsal nerve root, pass to the superior cervical ganglion, thence in the carotid or cavernous plexus to the Gasserian ganglion of the fifth nerve, and thence by way of the first branch of the fifth nerve and the long ciliary nerves to the iris. The cilio-spinal centre in the spinal cord receives impulses from many directions, any one of which is capable of producing a dilatation of the pupil. Thus irritating impulses coming in through the lower cervical and upper dorsal posterior spinal nerve roots from the skin of the neck, or if of sufficient intensity from any region of the body, can cause such a dilatation. It is possible that the total absence of such impulses when the posterior nerve roots in the cervical cord are injured causes the permanent contraction of the pupil, called spinal myosis. Impulses also reach the cilio-spinal centre from the cortex of the brain and from many subcortical centres in the basal ganglia and medulla. Attention directed to a distant object, with voluntary fixation of the eyes, or emotional changes, fear or anger, or any irritation of the medulla which excites the respiratory and vasomotor centres may cause dilatation of the pupil. It is the last-named cause which produces a contracted pupil in meningitis and cerebral disease. A general lowering of the nervous condition such as accompanies neurasthenia, hysteria, epilepsy, and anæmia is usually attended by a dilatation of the pupil. The exact path from the optic centres to the spinal centre is as yet undiscovered. A permanent loss of power of dilatation of the pupil in darkness is known to occur only when the cilio-spinal centre in the cord or the sympathetic nerve in the neck or its branches to the dilator pupillæ are destroyed by disease. This occurs in tabes, in paresis, in multiple sclerosis, and in bulbar palsy, and in myelitis of this locality. The hemiopic pupillary reflex of Wernicke has been described on page 560. When a patient presents anomalies in the action of the pupils the possibility of his being under the influence of some drug which affects their action must be considered before a conclusion is reached that a local or general disease of the nervous system is present.

Argyll-Robertson Pupil. This is a condition of loss of reflex of the pupil to light, while its action in accommodation is preserved. (See pages 298 and 653.)

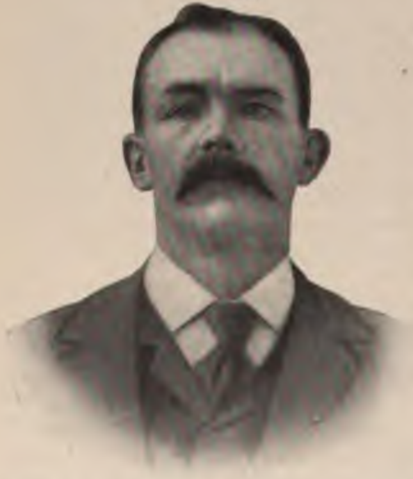
Paralysis of the Fourth Nerve. Patheticus Paralysis.

Paralysis of the fourth nerve is extremely rare and is only to be detected by a careful examination for double images with a red glass over one eye. The upright image is then seen to be upright by the normal eye, but appears to be displaced outward and turned obliquely by the affected eye. The fourth nerve supplies the superior oblique muscle and is of all the nerves the one best protected within the brain. It arises from a small group of cells beneath the aqueduct of Sylvius, and decussates with its fellow in the roof of the aqueduct just beneath the corpora quadrigemina posterior, and makes its exit immediately after its decussation on the dorsal surface of the crus lying upon the superior peduncle of the cerebellum. (Fig. 251.) It then curves around the crus lying upon the upper border of the pons Varolii, and so passes forward upon the side of the optic tract and enters the orbit through the sphenoidal fissure. Any of the causes which produce paralysis of the third nerve may cause paralysis of the fourth. Isolated paralysis of the fourth nerve without affection of the third or sixth is an important symptom of tumor of the cerebellum or of exudation on the under surface of the anterior lobe of the cerebellum. In a patient seen in Meynert's clinic at Vienna, who presented many symptoms of cerebellar disease, the existence of an isolated fourth nerve paralysis made it probable that the cerebellar lesion lay in the anterior and ventral part of the cerebellum, a diagnosis which was confirmed by autopsy.

Paralysis of the Sixth Nerve. Abducens Paralysis.

The symptom produced by paralysis of the sixth nerve is internal strabismus. This nerve supplies the external rectus, and when it is paralyzed the internal rectus, being unopposed, turns the eye inward, producing strabismus and an inability to turn the eye outward. Double vision always accompanies the strabismus, and the pupil is usually somewhat contracted on account of the position of the eyeball, but, of course, reacts to light. The sixth nerve arises from a group of cells upon the floor of the fourth ventricle, passes ventrally through the pons Varolii, making its exit in the groove between the pons and medulla, and then passes forward beneath the pons to enter the orbit through the sphenoidal fissure. As it has the longest course of any cranial nerve, disease at any part of the base of the brain is liable to involve it, hence abducens paralysis is a frequent symptom of intracranial disease, such as basilar meningitis, syphilis of the base, tumors of the brain, and fractures of the base of the skull. The same causes which produce oculomotor paralysis may cause abducens paralysis, and it is equally inaccessible to any treatment. The sixth nerve nucleus and the root of the nerve are often involved in disease of the pons Varolii. (See page 475.)

FIG. 255.



Paralysis of the left sixth, seventh, and eighth nerves from a fracture at the base of the skull.

The diagnosis between an affection of the abducens, due to disease in the nerve trunk or due to disease in the brain, can only be made from the presence of other symptoms. Paralysis of the face on the same side as the ocular palsy, and of the arm and legs upon the opposite side, indicates a lesion in the pons or on the base, producing unilateral pressure on the pons. In the patient whose face is shown in Fig. 255 the paralysis of the sixth nerve was associated with paralysis of the seventh and eighth nerves on the same side. This patient had suffered from a fracture of the base of the skull, but had recovered from all the symptoms excepting those present in these three nerves.

The Conjugate Motions of the Eyes. In all acts of vision the eyes move together, either being converged to a near object, diverged in looking at a distance, or turned together in any direction.

In convergence the internal recti act together and there is a contraction of the pupil.

In divergence the external recti act together and there is a dilatation of the pupil.

In conjugate lateral motions opposite muscles in the two eyes act together. In motions upward and downward homogeneous muscles act together. All these movements are primarily reflex and automatic, the sensations of objects upon the retina causing the appropriate action. For such acts the interaction of the various nuclei of the optic and motor nerves of the eyeballs is all that is necessary. And the very complete system of association fibres joining these various nuclei to one another provides for such combinations of movement. Lesions which destroy these association fibres, especially lesions in the posterior longitudinal bundle, interfere with conjugate lateral motions. Lesions in the nuclei themselves necessarily suspend consensual motions in any direction, both reflex and voluntary. The connections of these nuclei are shown in Fig. 251.

Conjugate movements may also be voluntary in origin. The motor centres in the cerebral cortex for ocular movements are located in the anterior part of the motor zone. (See page 422.) They send their impulses down to the subcortical centres by a tract which passes through the knee of the internal capsule, occupies a position near the median line in the foot of the crus cerebri and decussates in the raphé before it ends in the oculomotor nuclei. The tract conveying impulses of conjugate lateral motion passes directly to the sixth nerve nuclei, and thence an impulse is sent back to the opposite third nerve by way of the posterior longitudinal bundle. Fig. 256 shows this tract and demonstrates the fact that lesions at different parts of it cause paralysis of lateral conjugate motion of the two eyes.

Irritation of the motor centres in the cortex causes involuntary turning of the eyes away from the side of the lesion. This happens in epilepsy when the focus of irritation which starts the fit is unilateral. Paralysis of the motor centres in the cortex, or destruction of the tract which conveys their impulses will eventually result in conjugate deviation of the eyes toward the side of the lesion, for the eyes being once turned in that direction remain fixed, there being no possibility of a voluntary correction of this position. Hence in severe attacks of apoplexy when this tract is destroyed the patient is said to look toward his lesion.

The diagnosis of the situation of a lesion producing forced conjugate deviation of the eyes can only be made from the existence of other symptoms of cerebral or pons disease. These symptoms have been more fully discussed in Chapter XXIV.

Rheumatic Ocular Palsy. A paralysis affecting one or more of the ocular muscles must not be too hastily ascribed to a lesion of the nerve. These muscles are subject to rheumatic affections like other muscles, and occasionally a supposed ophthalmoplegia turns out to be a rheumatic myositis, and subsides promptly. The external rectus is the muscle most frequently affected.

FIG. 256.

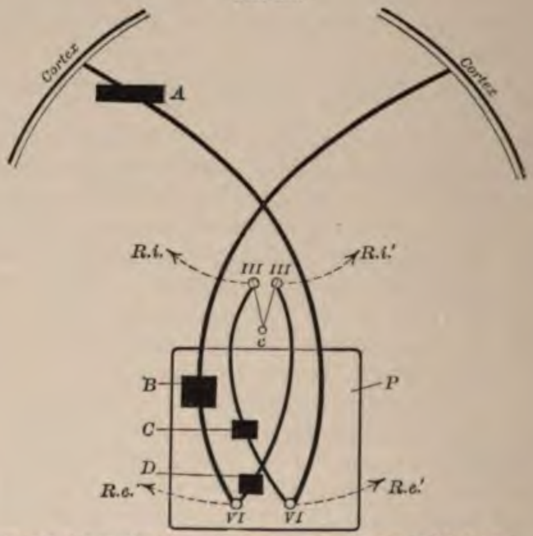


Diagram to illustrate the mechanism of conjugate movements of the eyes. *P.* Pons Varolii. *III.* Oculomotor nuclei. *VI.* Abducens nuclei. *Ri.* Rectus internus. *Re.* Rectus externus. A tract from the cortex passes to *VI.* A tract connects each *VI* with the opposite *III.* *C.* Centre of convergent movement of both eyes—a part of *III.* *A.* Lesion in the left half of the brain, causing a loss of voluntary turning of both eyes to the right. *B.* Lesion in the left half of the pons, causing a loss of voluntary turning of both eyes to the left. *C.* Lesion in the left posterior longitudinal bundle, causing a loss of voluntary conjugate motion of the eyes to the right. *D.* Lesion in the left *VI* nucleus, causing a loss of voluntary turning of the eyes to the left.

Recurrent Oculomotor Palsy. A rare condition consisting of combined paralysis of several of the muscles of temporary duration, but commonly recurring several times in the course of a short attack has been described and named recurrent oculomotor palsy. The oculomotor nerve is the one usually affected, but the fourth and sixth may also be involved. The attacks of double vision are attended by headache, nausea, vomiting, and by a fever, and occasionally by conjunctivitis. They have been termed ophthalmic migraine by Charcot. In one case seen by me the cause was probably malarial infection and the attacks ceased under quinine. Darquier[1] and Knapp[2] have collected and analyzed about forty cases. Autopsies are wanting to establish the lesion, which is probably a vascular one, congestion or œdema, either in the nuclei or in the nerve trunks.

Ophthalmoplegia.

Disease in the nuclei of the nerves that supply the muscles of the eyeballs and paralyze them is termed ophthalmoplegia. It has been divided into ophthalmoplegia interna and externa, according to the muscles affected. In ophthalmoplegia interna the action of the pupil is affected. In ophthalmoplegia externa the motion of the eyeballs and of the levator palpebræ are impaired.

Ophthalmoplegia Interna. Ophthalmoplegia interna is a very rare condition. It may be unilateral or bilateral. It may be acute or chronic in its onset. There are no cases with autopsy on record and the condition as a symptom is a curiosity.

Loss of reflex to light while reflex action in accommodation is preserved, called the Argyll-Robertson pupil, or reflex iridoplegia, occurs as a symptom in tabes, in paresis, in disseminated sclerosis, in softening or atrophy of the brain, in syphilis of the brain, in hydrocephalus, in tumors of the third ventricle or adjacent basal ganglia, and as a congenital condition. It is supposed to point to a suspension of action in the association neurones between the optic terminals and the oculomotor ciliary nuclei. These axones pass through the gray wall of the aqueduct of Sylvius, and in all these diseases except tabes microscopic lesions in this region have been observed. There is as yet no satisfactory explanation for the occurrence of this symptom in tabes. The condition is usually eventually bilateral, but may be unilateral at first. Loss of action of the pupil in accommodation is known to occur without loss of action to light. This is a rare condition, is usually unilateral and due to a lesion in the cervical sympathetic.

Ophthalmoplegia Externa. This term is applied to any form of paralysis of the muscles moving the eyeball, not due to a lesion in the periphery of one nerve alone. It is usually bilateral. It is usually partial, a few only of the ocular muscles being involved. It may, however, become total, under which circumstances the eyeballs are motionless. Inasmuch as the muscles affected are numerous, almost every possible combination of paralysis of different muscles has been observed. It is not, therefore, necessary to specify the different position of the eyeballs possible and the different forms of double vision possible in ophthalmoplegia.

Etiology. The symptoms may be produced either by lesions involving the roots of the oculomotor nerves in their passage through the cerebral axis, or by lesions limited to their nuclei. Lesions involving the roots are very common. They are due, as a rule, to hemorrhage from, or thrombosis or embolism in the small arteries, and especially in their terminal branches within the crus cerebri. These arteries enter through the posterior perforated space, and are terminal arteries. Tumors, spots of sclerosis, or syphilitic exudations in this region cause the same effect. The majority of cases on record of ophthalmoplegia externa which have been examined pathologically have been produced in this manner. Such lesions necessarily cause a destruction of other nerve tracts passing through the cerebral axis in addition to the oculomotor roots. It is from the presence of symptoms of such destruction that the pathological diagnosis is to be reached. Thus hemianæsthesia, hemiataxia, or hemiplegia of various types have been observed in association with partial ophthalmoplegia. And such an affection of the sensory and motor tracts in the cerebral axis may be bilateral, causing very extensive and complex groups of symptoms of which the oculomotor palsies may be the least noticeable, though they may be the most important as a guide to the localization of the lesion. (See Figs. 178 to 181.)

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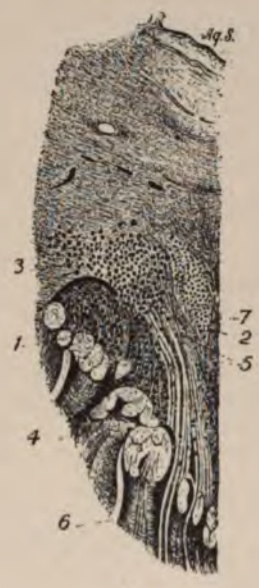
[1] Annales d'oculistique, Oct., 1893, tome cx. p. 258.

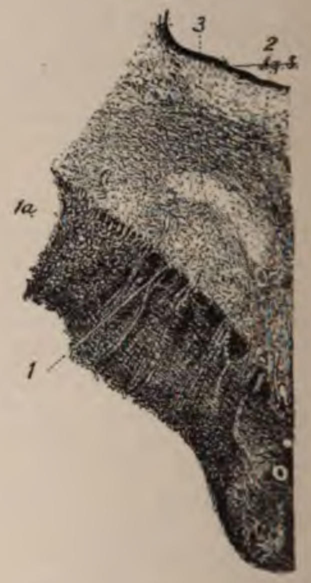
[2] Boston Med. and Surg. Jour., Sept., 1894.

FIG. 257.



Normal oculomotor nucleus. 1. Ventral nucleus. 2. Median nucleus. 3. Lateral nucleus. 4. Posterior longitudinal bundle. 5, 6. Nerve roots. 7. Raphé.

FIG. 258.



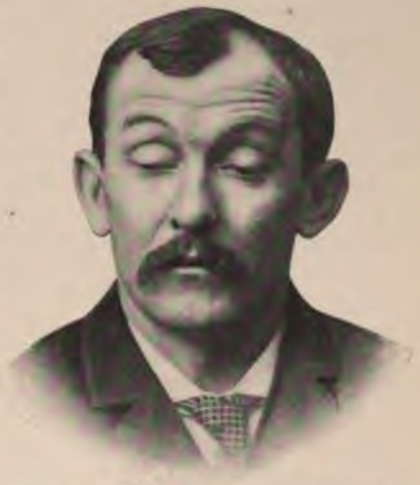
Atrophy of oculomotor nucleus. 1. Ventral nucleus. 1a. Dorsal nucleus. 2, 3. Edinger-Westphal nucleus. (Siemerling.)

Lesions involving the nuclei of the oculomotor nerves may be either acute or chronic, and resemble in character the lesions of anterior poliomyelitis.

Pathology. Acute ophthalmoplegia externa may occur from hemorrhage in the nuclei, from thrombosis or embolism in terminal vessels supplying the nuclei, or from inflammatory processes, probably infectious in origin, in the nuclei. It may also be the result of poisoning by bad food. Wernicke has named this condition polioencephalitis acuta. (See page 544.) The lesion found is a congestion of the vessels, exudation from them of small cells and leucocytes and serum, swelling and degeneration of all possible degrees in the motor cells, with destruction of their dendrites and axones, and consequent degeneration in the nerve roots and nerves. This lesion may be limited to the oculomotor nuclei, or it may be extensive in the cerebral axis, affecting other cranial nerves, in which case the ophthalmoplegia is only a part of an acute bulbar paralysis. A chronic atrophy of the nuclei has also been described and is shown in Fig. 258. These lesions resemble in every particular the lesions of anterior poliomyelitis.

Symptoms. The onset of *acute ophthalmoplegia* is usually sudden, with severe cerebral symptoms, vertigo, vomiting, headache, and even delirium and coma. The patient is always somnolent for some days, and occasionally develops an optic neuritis as well as the ocular palsy. In a few cases other symptoms of bulbar paralysis have developed, and a fatal result followed. In many the general symptoms subside in the course of two weeks, and the only permanent symptoms are the irregular ocular paralyses. The affection may be arrested in its progress, but usually leaves a chronic condition of paralysis. I have seen a case in which, after six months of steady improvement, complete recovery, lasting for fifteen years, ensued.

FIG. 259.



Patient suffering from chronic ophthalmoplegia externa. The wrinkling of the forehead in the effort to open the eyes is noticeable. The external strabismus can be seen.

Chronic ophthalmoplegia externa may be the terminal result of an acute attack, or it may be a primary condition. In a number of cases it has been a congenital condition. If primary it begins gradually and makes slow but steady progress, one muscle after another being affected. It may vary in intensity, some muscles recovering as others are involved. Fig. 259 shows the appearance presented by a patient suffering from this disease. In this case the disease has remained stationary for six years. It may even go on to recovery, the muscles gradually regaining their power. Such recovery occurs in the minority of the cases. In the cases which recover it is probable that the degeneration is only slight in degree, sufficient to arrest the function of the cells for a time. In this condition also a general chronic bulbar paralysis may eventually develop. It is not uncommon to find some defective action of the orbicularis palpebrarum and frontalis muscles supplied by the facial nerve, associated with ptosis; or some affection of the muscles of mastication. It is rare for bulbar palsy to advance to the oculomotor nerves, though it is not rare for it to develop subsequently to their paralysis. This is probably because a fatal result is reached early in bulbar palsy. As a curious confirmation of the statement that chronic ophthalmoplegia is quite homologous to chronic anterior poliomyelitis, the case of Kalicher may be cited in which, in the course of six months, the paralysis extended from the eyes to the face, and then to the arms and to the legs until total paralysis was present. The lesion in this case was chiefly vascular, the vessel walls being thickened, showing fatty degeneration and unusual dilatations, and hemorrhagic extravasation being found everywhere in the gray matter. Chronic ophthalmoplegia may be the first symptom of tumor in the corpora quadrigemina or of multiple sclerosis.

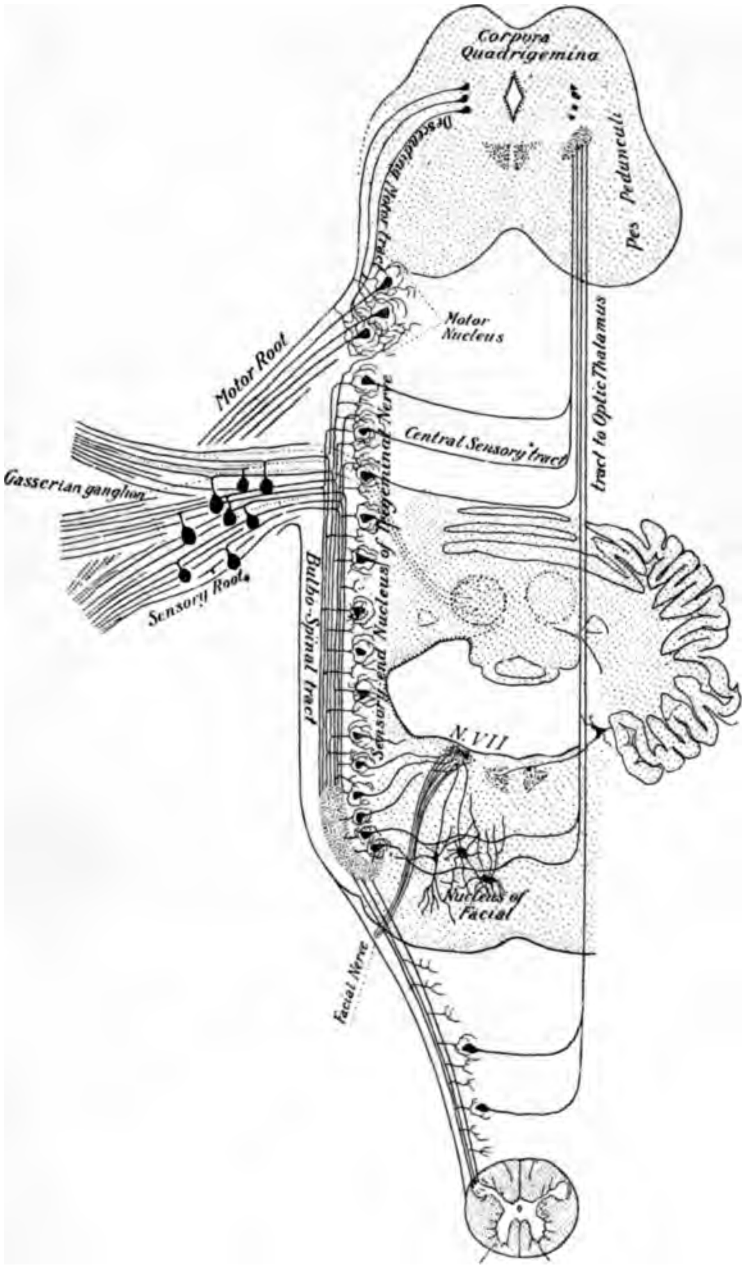
Prognosis. The prognosis in partial ophthalmoplegia externa is not as unfavorable as might be supposed. Over one-half of the patients recover. This seems to show that in the majority of cases the lesion is a vascular one and does not cause a degeneration of the nuclei. In the total chronic cases the prognosis is bad.

Treatment. The treatment of acute ophthalmoplegia is by the use of ice to the back of the neck, aconite, and phenacetin, and the administration of hot baths daily to produce free sweating. At the same time brisk laxatives and diuretics are to be used, and if the patient is plethoric leeches to the temples or behind the ears are to be applied. The eyes should be kept in perfect rest. In chronic cases it is best to correct diseases of the bloodvessels by the use of heart stimulants, nitroglycerin, and iodide of potassium, and later to stimulate the nerves by the use of strychnine. In cases where syphilis has been a possible cause a mercurial treatment, followed by the free use of iodide, is to be prescribed.

THE FIFTH NERVE: TRIGEMINAL NERVE.

Anatomy. The trigeminal nerve is made up of two parts, a motor and a sensory portion, the latter being much larger than the former. The motor part arises from a group of cells lying in the upper part of the pons Varolii in the lateral portion of the formatio reticularis. (Fig. 260.) It passes outward through the formatio reticularis, joins the sensory root, lies under the Gasserian ganglion, and leaves the skull with the inferior maxillary branch. It passes to the muscles of mastication, viz., the masseters, temporals, zygomatic, digastric, and myelohyoid muscles, which close the mouth and move the lower

FIG. 260.



Scheme of the neurones making up the fifth or trigeminal nerve. (Edinger.)

jaw. It also sends a filament to the tensor tympani muscle. The sensory part of the nerve develops from the neurones in the Gasserian ganglion. This large ganglion lies upon the nerve at the side of the pons (Fig. 245) upon the base of the skull. It has an extensive distribution upon the face and in the head, as shown in Plate XXIII., and all sensations from these parts come in through the nerve. The various areas of the skin of the face connected with the three great divisions are shown in Plate V. Its terminal central axones enter the pons from the ganglion and terminate in a long column of gray matter which is identical in its structure with the substantia gelatinosa of the spinal cord, and lies in the lateral part of the formatio reticularis, from the upper part of the pons to the lowest part of the medulla, where it is continuous with the posterior horn of the spinal cord. As these axones enter the pons a few bifurcate, but the majority turn downward, and some pass all the way down to the lowest level of the medulla, lying at the side of the column of gray matter just mentioned. Hence in cross-sections of the pons and medulla this root of the fifth nerve is cut across and appears as a semilunar-shaped white tract. (Fig. 181.) It should be called the descending root, as sensory impulses coming in pass down through it to reach the gray matter. But as this cranial nerve was formerly described as arising in the medulla and pons, like the motor nerves, it was originally named the ascending root of the fifth. The axones end in bushes within the substantia gelatinosa, and thence new sensory filaments arise which pass upward to the cortex. These appear to decussate in the upper part of the pons, as unilateral lesions of the pons below the entrance of the fifth nerve produce anæsthesia of the face on the side of the lesion only. They then join the general sensory tract in the lemniscus and formatio reticularis and pass to the cortex in the lower third of the sensory motor area. (See page 446.)

There is a long semilunar-shaped group of cells which lies on the lateral border of the gray matter lining the aqueduct of Sylvius, in the substance of the tegmentum, and extends for four millimetres beneath the corpora quadrigemina posterior. These cells resemble the cells of the column of Clarke in the spinal cord and are large and flask-shaped. They send axones downward in a long descending nerve root which is semilunar in cross-section and which enters the fifth nerve root opposite its exit at the level of the motor nucleus of the nerve. Some authorities[1] ascribe motor functions to this group of cells and root, though they were found to be normal in a case where paralysis of the muscles of mastication was present and where the motor nucleus of the nerve was degenerated. Other authorities ascribe trophic functions to this group of cells and root, and affirm that they are diseased in cases of hemiatrophy of the face. This statement is made by Mendel[2] on the basis of one case only. Others still regard them as a part of the sensory root.

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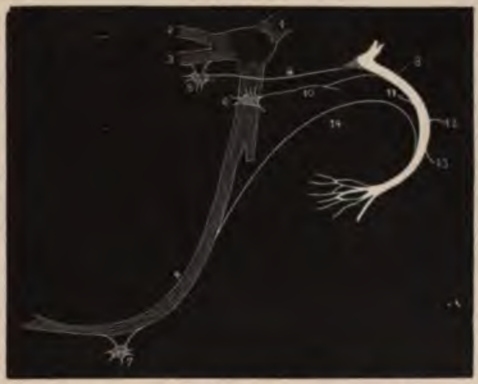
[1] W. A. Turner. Allbutt's System of Medicine, vol. vi. p. 787.

[2] Neurol. Centralblatt, vol. vii. p. 14.

The sense of taste is a part of the function of the fifth nerve, for complete division of this nerve behind the Gasserian ganglion suspends this sense. The nerves through which this sense is conveyed are two: 1. The anterior two-thirds of the tongue sends sensations of taste through the lingual nerves and chorda tympani, which passes into the Fallopian canal in association with the facial nerve, but leaves it at the geniculate ganglion, thence passes by the Vidian or great superficial petrosal nerve to Meckel's ganglion (sphenopalatine), and thence to the superior maxillary nerve, and thus into the second trunk of the trigeminal. 2. The posterior third of the tongue sends sensations of taste through the peripheral branches of the glossopharyngeal nerve, thence through the small superficial petrosal nerve or nerve of Jacobson to the otic ganglion, and thence by the inferior maxillary nerve into the third trunk of the trigeminal. (See Fig. 261.)

FIG. 261.



The facial nerve and its connections, within the aqueduct of Fallopius. 1. Fifth nerve, with the Gasserian ganglion. 2. Ophthalmic division of the fifth nerve 3. Superior maxillary division of the fifth nerve. 4. Lingual nerve. 5. Sphenopalatine ganglion. 6. Otic ganglion. 7. Submaxillary ganglion. 8. Facial nerve in the aqueduct of Fallopius. 9. Great superficial petrosal nerve. 10. Small superficial petrosal nerve. 11. Stapedius branch of facial nerve. 12. Branch of communication with pneumogastric nerve. 13. Branch of communication with glossopharyngeal nerve. 14. Chorda tympani. (Dalton.)

The protected situation of all these nerves accounts for the rarity of loss of the sense of taste. I have seen it once, however, in a case of severe chronic trigeminal neuralgia and anæsthesia probably due to destructive lesion of the root of the nerve. I have also observed a total loss of taste on one side of the tongue and pharynx after division of the roots of the nerve and after excision of the Gasserian ganglion done for the relief of neuralgia in several patients.

Paralysis of the Fifth or Trigeminal Nerve.

A lesion of the motor portion of the trigeminal nerve causes paralysis of the muscles of chewing. The lower jaw falls open in such cases, and hence both speech and swallowing are interfered with.

The patient usually supports the jaw with the hand, and thus assists both acts. It is to be remembered, however, that these muscles act symmetrically on both sides and together, and hence the destruction of one motor branch of the nerve does not cause very great disability. A little branch from the motor portion passes to the tensor tympani through the otic ganglion, and hence, when the nerve is paralyzed, the ear drum cannot be stretched, and consequently deafness appears. This may be accompanied by tinnitus aurium. When the muscles are paralyzed a gradual atrophy may occur which leads to a sinking of the temple and a thinness of the face, over the muscles which are paralyzed, and at the same time reaction of degeneration may appear in these muscles. Müller and Schultze both describe a slight difficulty in swallowing due to a paralysis of the muscles about the hard palate as having occurred in two cases. In both these cases there was a slight deviation of the uvula toward the paralyzed side. These symptoms were referred to a paralysis of the spheno-stapedius muscle, which receives a branch from the motor portion of the trigeminal nerve.

These symptoms of paralysis of the fifth nerve are of some interest, as the nerve is occasionally divided by surgeons in the operation for trigeminal neuralgia, and it is chiefly in such cases that the symptoms hitherto described have appeared. It is needless to add that in such cases the motor branch of the trigeminal nerve should be carefully avoided by the surgeon, so that the paralytic symptoms may be prevented.

Trismus or tonic spasm of the muscles of mastication is usually an affection associated with general convulsions or is a symptom of the infection of tetanus or lockjaw. It is not to be regarded as a local disease of the motor branch of the fifth nerve. Occasionally a spasm of these muscles upon one side of the body occurs in connection with facial spasm, and may be regarded as a local affection quite similar in its origin to blepharospasm and facial tic. Under these circumstances it is usually a reflex manifestation of some sensory disturbance about the jaw or throat, and until the origin of such reflex irritation is ascertained and removed treatment will not be successful.

Tonic spasm of the masseters has been known to occur after long-continued stretching of the muscles, as in a case reported by Peterson, where it developed after a long dental operation. It has been known to occur after a condition of paralysis in the muscles, as in a case reported by McConnell. Occasionally a fixation of the jaw and inability to open the mouth occur as a symptom in hysteria.

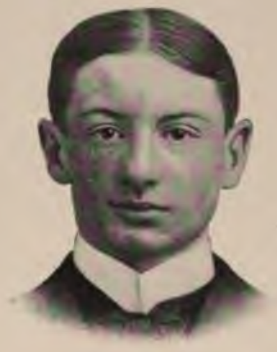
Clonic spasms of the muscles of mastication occur, causing a chattering of the teeth, as in a malarial chill or in an extreme condition of fear, or a grating of the teeth if the spasm is more slow and forcible. It occurs occasionally in paralysis agitans and in old age. Even in health every one has bitten the tongue or bitten the cheek unintentionally by an unexpected movement of the jaw. The disease is of no moment excepting in children, when grinding of the teeth at night often causes much annoyance to the parents and sometimes disturbs the child's sleep. This may be due to any disease which produces an irritation of the nervous system, such as irritation of the intestine by worms, irritation of the genitals by smegma, irritation of the throat by adenoids, or irritation of the ear by wax. Occasionally grinding of the teeth is an early sign of irritation upon the base of the brain by a beginning basilar or tubercular meningitis, and in many cases of this disease the symptom appears at some time in the progress of the case. It has been known to occur as a symptom of tumor of the base of the brain. The spasm is uniformly bilateral. In the vast majority of children's cases the symptom is outgrown, and it should not cause any alarm excepting in connection with other symptoms of tetanus or of tubercular meningitis.

All these forms of facial spasm are to be treated as in facial tic. (See page 670.)

Bernhardt has recorded one case of congenital paralysis of the trigeminal nerve which was accompanied by abducens and facial palsy, and this appears to be a unique case in the literature. Injuries of the fifth nerve upon the base of the brain by tumors, syphilitic or tubercular exudations in the meninges, or fractures of the base are very rare. When the Gasserian ganglion is involved herpes zoster upon the face may appear. A neuritis of the trigeminal nerve following cold has not been described, yet it is not at all unlikely that very many cases of trifacial neuralgia (see page 82) are really due to this cause. Hemorrhage or softening in the pons varolii or medulla at any point in the fifth nerve nucleus, or tumors, or patches of sclerosis in the same locality may cause paralysis of the nerve. Thus the symptoms may be present in any form of gross lesion in the cerebral axis. It also appears as a symptom in some cases of bulbar paralysis. In these cases the existence of other symptoms of bulbar disease will enable a diagnosis of the site of the lesion to be reached. It has also been observed in syringomyelia when the gliomatous degeneration extended into the pons.

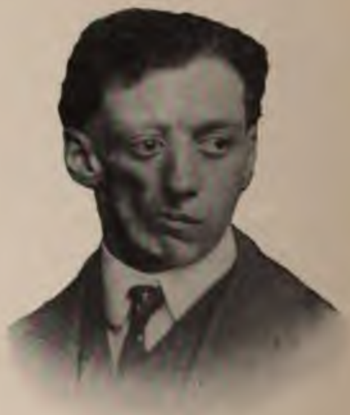
The symptoms of paralysis of the sensory portion of the trigeminal nerve are numbness and anæsthesia of the face. In Plate XXII. the exit of the three great branches of the sensory portion upon the face are shown, and in Plate V. their distribution to the skin is shown. The first branch supplies the tear ducts and glands, and any loss of its function is followed by a dryness of the eye. The second branch controls the sensation of the mucous membrane of the nose and mouth, and dryness of these parts accompanies this injury. The second and third branches contain all the fibres concerned in taste, and hence lesion of these parts results in the loss of this sense. The vasomotor and trophic functions of the fifth nerve are important, and trophic symptoms are exceedingly common in cases of any injury of the fifth nerve. Whether these are due to the suspension of sensation may be a matter of discussion. They consist of ulcerations of the cornea, falling of the teeth, changes in the hard gums and in the mucous membrane of the mouth and nose, with undue dryness and tendency to ulceration and the formation of crusts, and also the development of herpes zoster, which may occur in the nasal and lingual cavities as well as on the skin. The fifth nerve supplies the tongue with sensation as well as with taste, and unnoticed injuries of the tongue in chewing are not uncommon when the fifth nerve is anæsthetic. A loss of the reflexes of the face occurs in paralysis of the fifth nerve, so that winking does not occur when the eye is irritated; tears do not flow, sneezing cannot be produced by inhalation of irritating substances or by tickling of the nose. The sense of smell is somewhat impaired, partly on account of the dryness of the mucous membrane of the nose, and partly because many irritating substances like ammonia, alcohol, and substances which irritate the mucous membrane are really perceived through the trigeminal nerve. It is possible that the secretion of saliva and swallowing upon the paralyzed side may be suspended.

FIG. 262.



Hemiatrophy of the face. The condition developed slowly during five years, and then came to a standstill at the point shown.

FIG. 263.



Hemiatrophy of the face. The condition had developed in two years. (Kindness of Dr. Mailhouse.)

The common form of disease of the fifth nerve is trigeminal neuralgia. This has been described on page 82 *et seq.* It is there shown that such neuralgia is usually due to a lesion of the Gasserian ganglion.

Hemiatrophy of the Face. A gradually advancing atrophy of one-half of the face has been observed. It is a very rare affection. It is characterized by a very slowly progressive atrophy affecting the skin, the subcutaneous fat, the muscles, and the bones in about equal degree. As a rule, this condition develops in youth; it has not been observed before the age of ten years, and is rare after the age of twenty years. For some time it may escape notice, but gradually the asymmetry of the face becomes more and more evident, the skin is manifestly thin, and is often slightly pigmented, the cheek sinks in upon the affected side, the temple also sinks, and when the disease is thoroughly developed, as in the case shown in Fig. 263, palpation reveals the decided atrophy of all the tissues. Sometimes the hair on the affected side falls out, but this is not a uniform result. Sensibility is in no way affected and the muscles are not paralyzed, although as they become thin they are weaker than those of the healthy side. After several years the condition appears to come to a standstill, and the disease does not lead to a fatal termination. Occasionally the disease makes rapid progress, as in the patient shown in Fig. 263. In this man the disease had been in progress only two years. He suffered from spasms of the masseters and much pain in the face, but had no anæsthesia and no other symptoms of intracranial disease. The pathology of this disease is unknown. There is no known treatment.

THE SEVENTH NERVE: THE FACIAL NERVE.

The facial nerve is the great motor nerve of the face. It arises from a large nucleus which lies deep in the formatio reticularis of the pons varolii. (Fig. 198.) From this nucleus the fibres pass backward and inward toward the floor of the ventricle, curve about the nucleus of the sixth nerve, forming an angle known as the knee of the facial, and then turn outward, traverse again the formatio reticularis of the pons, and make their exit in the groove between pons and medulla in close proximity to the eighth nerve. It then enters the internal auditory foramen of the petrous portion of the temporal bone and, after passing through the bony canal called the aqueduct of Fallopius, issues from the stylomastoid foramen upon the face just under the lobe of the ear. It there divides into many branches which supply the various muscles of the face. (Plate XXII.) Just before entering the auditory foramen a ganglion is seen to lie on the nerve. This is the geniculate ganglion. As motor nerves do not possess such ganglia, it is evident that at this point some sensory fibres must be associated with the motor fibres. These really are sensory fibres belonging to the chorda tympani—a sensory nerve of taste which joins the seventh nerve in the Fallopian canal and leaves it at the ganglion to pass by way of the Vidian or superficial petrosal nerve to the superior maxillary branch of the fifth. Occasionally, when inflammatory processes attack the seventh nerve in the canal where this sensory nerve lies beside it, its fibres are involved, and then the sense of taste is lost on the anterior two-thirds of the tongue. A small nerve lies at the side of the facial between the ganglion and the pons and enters it between the seventh and eighth nerves. This is the nerve of Wrisberg. It is probably a sensory nerve. It passes toward the glossopharyngeal nucleus, where it ends. It has been thought to be a vestige of the extensive sensory facial branch found in fishes. Fig. 261 shows the various branches of the nerve in its course. The successive invasion of each branch from without inward by a progressive neuritis enables one to locate the process of a neuritis in the nerve, as will be indicated in the section on diagnosis. The facial nucleus is closely joined to many other cranial nerve nuclei by association fibres. One distinct branch from the oculomotor nucleus joins the facial nucleus and sends some filaments directly into the nerve trunk. Thus an anatomical basis for the many reflex and automatic acts in which the muscles of the head and face take part is secured. Thus in the acts of winking, breathing, in motions attendant upon sensations of sight, and sound, and taste, and smell, as well as in the various bodily sensations, changes of the facial expression are constantly seen which are automatic. Mental states are also reflected in the face unconsciously, and this is secured by an intimate association of the facial nuclei and the centres in the optic thalamus. Any disease in the pons, such as hemorrhage, softening, sclerosis, or tumor will suspend these reflex acts. The facial nucleus is also directly joined to the cerebral cortex, receiving impulses from the lower third of the motor area by a tract which traverses the knee of the internal capsule and lies in the median part of the motor tract in the crus cerebri. Its fibres leave the motor tract in the pons, pass backward in the raphé, where they partially decussate with those of the opposite side, and end in the nucleus. Thus each hemisphere is joined to both nuclei. Lesions in the course of this tract arrest voluntary movements of the face, but do not suspend the reflex acts. (See Figs. 173 and 174 and page 432.)

Facial Paralysis. **Pathology.** As facial paralysis usually recovers, but few pathological observations are available. Markowski[1] found in a case of facial palsy due to cold that the nerve was normal from the medulla to the geniculate ganglion, but from the ganglion downward into the periphery there was a well-marked degeneration. The neuritis was parenchymatous and not interstitial. The peripheral branches contained many fully degenerated fibres, a few remaining globules of myelin in a state of disintegration, and many little fat cells. Some of the branches contained normal as well as degenerated nerve fibres, and very few degenerated fibres were found in the superficial petrosal and stapedius nerves. The entire process was a pure degenerative neuritis without any participation of the neurilemma. A similar lesion was found by Darkschewitch and Tichonow.[2] Dejerine and Theohari[3] have examined a nerve after facial palsy and found a well-marked degeneration in all the branches of the nerve, though the lower branch was more affected than the others. There was no evidence of interstitial changes. While the root of the facial nerve showed no degeneration, examination of the nucleus by the Nissl stain demonstrated a disappearance of the fine Nissl bodies and a shining appearance such as is found in cells whose function is suspended. This was also seen by Flatau[1] in another case.

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[1] Arch. f. Psych., Bd. xxiii. p. 367.

[2] Neurol. Centralbl., 1893, p. 329.

[3] La Semaine Medicale, 1897, p. 453.

Etiology. While facial palsy due to maldevelopment in fœtal life has been occasionally described, the majority of congenital cases are due to pressure exerted upon the facial nerve during delivery. This pressure may be produced by forceps applied to the head or by a misplaced hand or shoulder pressing upon the face during labor. Bernhardt[2] has recorded a number of such cases, and every obstetrician has met with them. In some of these cases the orbicularis oris has not been paralyzed. Children occasionally suffer from facial paralysis following a blow on the ear or an attack of the mumps. Tubercular swelling of the glands of the neck, causing pressure on the nerve is an occasional cause of facial palsy in children, but the most common cause in children is otitis media producing an inflammation of the nerve in the Fallopian canal adjacent to the inflamed ear. This also is a cause of facial palsy in the adult in 7 per cent. of the cases. Intracranial disease, such as tumors of the brain, or exudations upon the base of the brain, or syphilitic inflammation of the nerve trunk within the cranium compressing the nerve, may produce paralysis. Fracture at the base of the skull and caries of the petrous portion of the temporal bone, involving the nerve in its course have also produced facial palsy. The usual cause of facial paralysis is supposed to be exposure to cold. Philip ascribed facial palsy to this cause in 72 per cent. of the cases. Remak[3] found this a cause in 45 per cent. of his cases, but Thomas[4] describes it as a cause in only one-quarter of his cases. In many of my cases a history of a draught upon the side of the face has been obtained, and it seems probable that this may produce such congestion of the nerve as to lead to facial paralysis. The disease does not occur more frequently in winter than in summer, but Bernhardt[5] found it more common in women than in men and more common in men who were shaven than in those who wore a beard. The Germans call all cases due to exposure to cold "rheumatic," but there appears to be no distinct relation between facial palsy and rheumatism. In very many cases no ostensible cause can be ascertained.

The most plausible explanation of the occurrence of facial palsy is that offered by Philip[6] who believes that a congenital narrowing of the stylomastoid foramen in certain persons predisposes them to a compression of the nerve upon any slight congestion of its trunk. It is probable that an unnaturally large nerve lying in a normal foramen might produce some such tendency. A certain number of cases of facial palsy develop during the night, when pressure upon the pillow, combined with venous stasis in the canal, might produce a pressure in such individuals. One attack of facial palsy predisposes to another, and in 7 per cent. of the cases there is a recurrence of the disease. Facial paralysis may accompany any form of multiple neuritis, and is then often bilateral. I have seen such cases in adults suffering from alcoholic multiple neuritis, also from severe lead palsy, and several cases in children after diphtheria.

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[1] Zeitsch. f. klin. Med., 1897.

[2] Berliner klin. Woch., 1899, No. 31.

[3] Remak. Neurol. Centralbl., 1899.

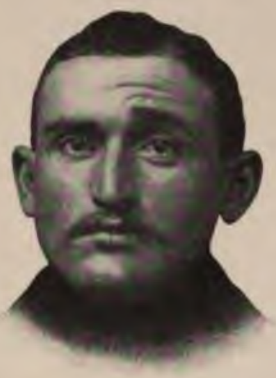
[4] Thomas. Jour. Amer. Med. Assoc., 1898, No. 21.

[5] Berliner klin. Woch., 1888, No. 7, and 1892, No. 30.

[6] Dissert., Bonn, 1890.

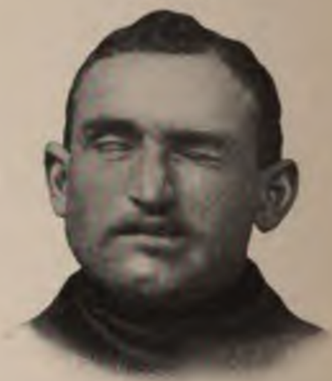
Symptoms. The symptoms of facial paralysis are very noticeable. There is a total paralysis of all the muscles upon one side of the face, including the forehead and the muscles which close the eye. The forehead is smooth, its wrinkles are no longer evident, and the eyebrow is immovable. Attempts at frowning are unsuccessful on the paralyzed side, the eye remaining open, and when the patient is told to close it he merely rolls the eyeball upward, leaving the sclera visible. He is not aware, however, that he has not closed the eye. The reflex act of winking is abolished, and the consequent exposure of the eyeball often leads to conjunctivitis. As the lower lid is not approximated to the eyeball, tears are no longer directed into the tear duct, and may run down upon the cheek. The nasolabial fold of the cheek is flat, the corner of the mouth hangs down, and there is a total lack of the normal play of facial expression during emotion or in conversation. The flat, expressionless countenance gives a mask-like appearance to the face and attracts attention at once. In the act of respiration the ala of the nose is not dilated and the cheek often flaps. All voluntary motions about the mouth are suspended, hence whistling, blowing, pursing of the lips, drinking, or moving the food about in the mouth during the act of chewing are imperfectly performed, and the pronunciation of labials is often indistinct. The patient often bites the cheek in the act of chewing, as the buccinator fails to act. Those who can move the ears lose this power. Occa-

FIG. 264.



Facial paralysis of the right side. Attempt to raise the eyebrows.

FIG. 265.



Facial paralysis of the right side. Attempt to close the eyes.

sionally, when the mouth is opened, the tongue appears to protrude toward the healthy side. This deviation is often apparent rather than real, the mouth being opened unevenly. If, however, the deviation of the tongue is real, it is due to a paralysis of the muscles attached to the hyoid bone, allowing this to fall upon the paralyzed side. Occasionally in facial palsy a deviation of the uvula has been noticed. One side of the uvula may be elevated, and it is then turned away from the paralyzed side, or it may fall toward the paralyzed side. This symptom is a rare one, and I have seen it only occasionally. While mentioned by Erb and others, it has been denied by Horsley and Beevor, who consider it a mere accident and not a symptom of facial palsy. It is quite certain that the motor innervation of the palate comes from the vago-accessorius. Schultze[1] has found such a deviation in but one case in 55. In some cases of facial palsy the sense of taste is affected upon the anterior two-thirds of the tongue upon the paralyzed side. This may be present, however, without attracting the notice of the patient.[2] It can be discovered by applying sweet, salty, or bitter solutions to the tongue with a fine camel's-hair brush or by passing a galvanic current through the tongue, when the galvanic taste is no longer produced. This symptom is only present when the lesion in the nerve is a deep one and involves that part of it within the Fallopian canal, where the chorda tympani joins it. An unusual acuteness of hearing and tinnitus aurium are occasionally complained of. They are due to the tension of the drum membrane consequent upon paralysis of the stapedius muscle. Under these circumstances the lesion is very deep in the Fallopian canal or upon the base of the brain. If the paralysis is an accompaniment of middle-ear disease, or if the auditory nerve is affected upon the base of the brain, where it lies in close apposition to the facial nerve, deafness and vertigo will be associated with paralysis. The patients sometimes complain of pain behind the ear at the time of the onset of the affection, and are often tender to pressure at the exit of the nerve. They sometimes have pain in the side of the neck, and occasionally herpes appears below the ear or on the side of the neck.

After the paralysis has been present for some little time stiffness in the muscles of the face is often felt, but it is rarely associated with neuralgic pain. If the case does not go on to recovery the facial muscles atrophy and contract, producing a very distressing sense of stiffness in the side of the face. This contracture of the muscles may restore the original lines of the face and the original facial expression, and may even elevate the corner of the mouth slightly, so that at the first glance the paralysis may be thought to be in the well side, but any attempt at voluntary movement will demonstrate the immobility of the truly paralyzed and contractured muscles.

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[1] Lehrbuch der Nervenkrankheiten, Bd. i. p. 5.

[2] Lermoyez, M. Annales des Mal. de l'Oreille, 1899, p. 564.

The electrical contractility in the facial muscles is usually altered. In some cases the faradic contractility is preserved and the galvanic contractility is slightly increased, so that the muscles respond to a weaker current than those upon the normal side. In these cases recovery occurs within three or four weeks. In cases of medium severity, however, there is a partial reaction of degeneration. The faradic contractility is very much diminished or even lost, the galvanic contractility is heightened, so that only a weak current is necessary to produce contractility, and the cathode closing contraction remains greater than the anode closing contraction. In the course of six weeks the galvanic contraction becomes normal; after two months the faradic contractility returns, and these patients recover in between two and three months. In severe and permanent cases there is a total reaction of degeneration, a complete loss of faradic contractility, and a gradual reduction in the galvanic contractility, so that very strong currents are necessary to produce any effect, and here the anode closing contraction is greater than the cathode closing contraction, and the opening contractions may be equivalent to the closure contractions. Such a reaction of degeneration may remain for a whole year, and even then recovery may ensue. In fact, Remak records a case of recovery after three years' continued reaction of degeneration. The electrical reactions, therefore, are important not only as symptoms but as prognostic indications in this affection.

The course of the case varies in different conditions in accordance with the severity of the initial lesion. In the majority of cases all symptoms develop within a few hours of the onset, and remain practically stationary for from two to three weeks, during which time no voluntary movement at all is possible. In the lighter cases the symptoms then gradually subside, and recovery results in four to six weeks. In more severe cases such recovery does not occur under six months. In the severer type a year may ensue before any relief is evident, and in a few cases no recovery occurs, the condition becoming permanent, the muscles becoming contractured, and very often being the seat of twitching and spasms which are annoying. It is in these severer cases that conjunctivitis, unless prevented by antiseptic alkaline lotions to the eye, occurs as a complication.

Diagnosis. The diagnosis of facial palsy presents no difficulties as the symptoms are very distinct. But facial paralysis may occur not only from a lesion of the nerve trunk, but from a lesion of the brain at any point in the motor tract between the cortex and the facial nerve nucleus. Facial paralysis produced by a cortical lesion or by a lesion in the motor tract above the level of the nucleus of the facial nerve is less complete than facial paralysis from lesion in the nerve, and is not attended by complete paralysis of the act of closing the eye. Winking is always possible, and the muscles of the forehead are less commonly involved. In emotional excitement the facial muscles act normally so that the play of expression occurs even though voluntary movement is suspended. In this form also there are no changes in the electrical contractility in the paralyzed muscles. While it is possible that an isolated facial paralysis from cortical or subcortical disease may occur, such paralysis is usually the accompaniment of hemiplegia, and hence is not likely to be mistaken for paralysis due to a lesion in the course of the trunk of the nerve.

Facial paralysis due to a lesion of the facial nucleus in the pons Varolii is almost uniformly attended by other symptoms of pons disease, such as (*a*) a paralysis of the sixth nerve, which lies adjacent to the seventh nerve nucleus; or (*b*) an implication of the motor or sensory tract passing to the opposite arm and leg, which pass close by the situation of the facial nucleus, thus producing alternating hemiplegia or hemianæsthesia (Fig. 177); or (*c*) the symptoms of bulbar paralysis; or (*d*) the symptoms of muscular dystrophy. Gowers has reported a case of isolated infantile palsy of the face analogous to infantile spinal paralysis. In this case the orbicularis oris escaped. The characteristics of the facial paralysis when the nucleus is affected are the same as those when the nerve trunk is diseased, but there is no affection of taste.

The diagnosis of disease upon the base of the brain between the exit of the facial nerve from the pons and its entrance into the internal auditory foramen can only be made from the presence of other symptoms of intracranial disease such as are due to pressure upon the side of the pons or to implication of other cranial nerves, notably the sixth or the eighth. The diagnosis of the location of the lesion in the course of the nerve within the Fallopian canal is not difficult. If the nerve is affected near the geniculate ganglion or prior to the giving off of the little branch to the stapedius muscle, tinnitus aurium and acuteness of hearing will be noticeable symptoms. They will not be present, however, if the lesion is nearer to the exit of the nerve than the point from which this branch arises. If the nerve is injured in the Fallopian canal between the point at which it is joined by the chorda tympani and the point at which this nerve is given off, a loss of taste in the anterior two-thirds of the tongue will be present. Such a loss of taste does not occur if the lesion is near to the exit of the nerve at the stylomastoid foramen or is in the face after its exit from this bony canal. (See Fig. 261.)

Prognosis. The prognosis in facial paralysis depends upon the cause and the severity of the attack. In lesions of the nucleus within the brain or of the trunk on the base of the brain, and in lesions of the nerve following otitis media, recovery is rare. When the cause is cold or is unknown, the majority of patients get well. The severity can be determined with a fair degree of accuracy by the electrical examination, as already described, and this examination often aids the prognosis.

Treatment. If the patient is seen within two or three days of the onset of the disease it is well to apply a small blister over the exit of the nerve beneath the ear. If the cause of the affection can be ascertained and can be removed, especially if it is otitis media, this should be done, but in the majority of cases it is impossible to ascertain a cause, and hence the treatment must be symptomatic. It should consist of massage of the face, done by the patient with the tips of his fingers frequently during the day, and this can be aided by placing one finger in the mouth and so compressing and kneading the muscles between the two fingers. The muscles should be treated with electricity daily, that current being used which produces a contraction. But it is not advisable to continue electrical treatment longer than six months, for in the cases which last beyond this time the electrical stimulation favors the development of contractures. When contractures occur in the chronic cases warm applications of water and massage may alleviate the sensation of stiffness. It is useless to apply the faradic current when the muscles do not respond to it. Iodide of potassium in five grain dose after meals and small doses of strychnine have been thought by some writers to hasten recovery.

Facial Spasm. (**Tic Convulsif.**) A spasmodic twitching of the muscles supplied by the facial nerve is not an uncommon affection. It is usually primary, and not secondary to facial paralysis. All the muscles may be affected by the spasm, or it may be limited to the muscles about the eye, the orbicularis palpebrarum, when it is called "blepharospasm." Usually the zygomatic muscles and the levator anguli oris are affected with the orbicularis palpebrarum. When this is the case the spasm usually extends to the muscles of the chin. It is a unilateral affection at first, but may become bilateral. The slight bilateral twitching of the face sometimes seen in childhood or in hysterical individuals is not included in tic convulsif.

Facial spasm is a disease of adult life, not usually developing until after the age of forty years. It occurs in women more frequently than in men and much more commonly in persons of a neuropathic constitution or in those who are subject to migraine, anæmia, or cachectic conditions. A sudden mental shock has been known to produce it.

A spasmodic contraction of the muscles anywhere in the body is rarely evidence of primary motor irritation. It is usually secondary to a sensory impulse, the contraction of the muscles being a response to such an impulse. Thus the act of winking is primarily due to an irritation of the conjunctiva, and blepharospasm can be usually traced either to some local disease in the eyelids, to some strain of the ocular muscles, or to some defect of vision. The most sudden or lightning-like contractions in the facial muscles are seen in connection with facial neuralgia, and it is probable that in the majority of cases of facial spasm the cause is to be found in some irritation in the domain of the trigeminal nerve Thus irritation in the nose, or about the teeth, or even in the mouth or throat, or in the scalp, is sufficient to produce a facial spasm, and many cases can only be relieved by the removing of the source of irritation. It is not impossible that small sensory filaments of the trigeminal nerve may be the seat of neuritis without any attendant pain, and that the result may be a facial tic. In the severe spasm of torticollis it is possible for an extension of the spasm to the facial muscles to occur, so that the spasm not only turns the head, but twists the face. Cases have been recorded of facial spasm due to a pressure of tumors on the facial nerve within the skull.[1] Facial spasm has also been observed in irritating lesions of the facial area in the motor cortex of the brain, but these spasms are rarely as constant, as frequent, as sudden, or as extreme as those in true facial tic. They are usually attended by spasms in the neck and arm and by other symptoms of cortical disease. Nevertheless, it is well to remember that a facial spasm may be the first sign of cortical epilepsy. (See page 422.)

Symptoms. The chief symptom of the affection is the sudden, lightning-like twitching of the muscles of the face. Such twitching occurs at intervals, or a series of twitches may occur rapidly, one after another, each paroxysm lasting several seconds, or even a minute. The final contraction may be a tonic one, so that the face is drawn up and is motionless for a few moments. Any attempt at looking fixedly at an object, or moving the face, or talking, or chewing is liable to be followed by a spasm, and sometimes a cold draught is sufficient to bring it on. Some patients complain that the spasm is set up by a strong light, under which circumstances it is probable that the irritation of the optic nerve is sufficient to set up a contraction. For this reason many patients protect the side of the face by a thick woollen pad, which keeps out light and cold. The spasm is never painful, and as there is no paralysis, voluntary motion is perfectly possible. There is no change in the electrical contractility of the facial muscles. In one case recorded by Keen[2] the spasm extended from the facial nerve to the muscles of mastication, the tongue, and muscles of the neck. Occasionally a tender spot in the region of the fifth nerve or slight areas of anæsthesia may be discovered by careful examination, a fact which supports the theory that the facial spasm is secondary to disease in the trigeminal nerve.

The course of the case is usually a very slow one. The spasm begins in a few muscles, and extends to others until the entire face is involved, and the disease may remain for many weeks or months, and then suddenly cease. It is particularly liable to return. Not infrequently there is no permanent recovery. The patient is liable to the spasm all his life. Hence the prognosis is unfavorable, especially if the disease has lasted more than a month.

Treatment. Treatment should consist in a very careful attempt to discover some source of reflex irritation by examination of the functions of the eye, careful investigation of the throat and nose and of the teeth; and the removal of such irritation if possible. In one patient whose entire left side of the face was in constant spasm examination showed that a marked defective visual power in the right eye had thrown all the work of vision on the left eye. This, too, was astigmatic and myopic. The proper adjustment of glasses resulted in a progressive cure, complete within a month, although the condition had been present for over a year. If no cause is found the disease must be treated symptomatically by hot applications to the face and by the free use of sedatives, of which bromide, cannabis indica, morphine, gelsemium, and conium are the most useful. Electricity is of no use. Counter-irritation by blisters or cautery also fails to relieve. Stretching of the facial nerve, which has been attempted, is usually followed by paralysis, but when this subsides the twitching returns.

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[1] Schultze. Virchow's Archiv, vol. lxv. p. 385. Vuss, Neurol. Centralbl., 1886.

[2] Transactions of the American Surgical Association, 1886.

Some cases of tic convulsif are of interest because of the possibility of locating the seat of irritation by means of hypodermic injections of cocaine. In two cases under my care the tic had been of long duration and had given the patients much discomfort and had failed to yield to any remedies. In neither case was there any localizable pain. Inasmuch as the majority of such cases of facial spasm are due to irritation in the course of the trigeminal nerve, it was necessary to determine, first, whether the spasms were reflex, and, second, if so, in which branch of the trigeminal nerve the irritation began. This was arrived at by injecting cocaine beneath the skin of the face successively at the point of emergence of the supraorbital, infraorbital, and dental branches of the trigeminal nerve. In the first case the injections had no effect on the spasm when the upper two branches were anæsthetized, but the injection at the mental foramen in the lower jaw was succeeded by immediate stoppage of the spasm for one-half hour, or until the local effect of the cocaine had subsided. This patient was then operated upon by Hartley, who exsected this branch of the trigeminal nerve at its exit from the jaw with the effect of permanent cure of the tic.

In the second case injection in the infraorbital branch of the nerve stopped the spasm, and here division of this branch was followed by permanent cure of the tic.

Sometimes a local spasm in the risorii may occur. Thus a well-nourished young woman, not anæmic, suffered from a bilateral spasm of the middle muscles of the face, so that she looked all the time as if smiling, and the upper lip was stretched tightly back across the upper teeth, and this impeded her speech, her eating, and gave her the appearance of laughing all the time, when she had no such desire. This condition came on very suddenly after a great mental shock, soon after the birth of her first baby, and persisted for six months. The chief discomfort lay in the great limitation of talking. There was no affection of the tongue or of the larynx, but all letters involving the use of the lips were imperfectly pronounced, especially when her attention was directed toward it. When her attention was diverted by an examination of her eyes and throat the spasm relaxed and the lip assumed its normal appearance, but the moment she began to speak, or the moment that she tried to smile, the sudden contracture of the muscles resulted in a flattening of the upper lip against the teeth, where it was held rigid. She appeared to be perfectly well in every other respect; there was a slight tendency to divergence of the eyes; there had never been any pain or difficulty in chewing. There was no pain on pressure along the fifth or seventh nerves; there was no apparent obstruction in the nose or throat. The condition still persists in spite of treatment.

THE EIGHTH NERVE: THE AUDITORY NERVE.

The eighth or auditory nerve consists of two distinct portions, the cochlear and vestibular nerves, which, though joined in the trunk, are separate from one another both in their peripheral termination and in their central nuclei.

I. The cochlear part of the nerve is the nerve of hearing. Like all nerves of special sense, it has a special mechanism in the periphery, the organ of Corti, which is a sort of natural harp, its strings vibrating to different tones, each string being in reality an epithelial cell connected with a nerve filament whose neurone body lies within the cochlea and sends a central filament into the auditory nerve. The terminations of this nerve in the nuclei of the medulla and their connections with the brain have already been described. (Page 458.)

Deafness. Deafness is usually due to some disease within the ear affecting the peripheral fibres of the nerve. But affections of the acoustic nerve produce disturbances of hearing.

Deafness due to disease of the cochlear fibres of the acoustic nerve is distinguished from deafness in the outer or middle ear by the loss of power of perceiving sound through the bones of the head. In a normal condition the sound of the tuning-fork can be heard both when held to the ear and when applied to the teeth, or forehead, or petrous portion of the temporal bone. The sound of the tuning-fork is normally heard longer when the vibrations are conveyed through the air than when they are conveyed through the bone. If there is disease of the middle ear, producing deafness, the tuning-fork is heard through the bone, but not when held near to the ear. If the deafness is due to disease of the auditory nerve the tuning-fork is no longer heard when applied to the bone, or is heard less distinctly than when held outside of the ear. In auditory nerve deafness the higher pitched sounds are less distinctly heard than low notes. This may be tested by the Galton whistle. In auditory nerve deafness hearing is not increased in a noise as it is when disease lies in the middle ear. In auditory nerve deafness inflation of the ear by Politzer's method does not improve the hearing.

In auditory nerve deafness changes to the electrical reactions are also present, which are not found in deafness from otitis media. These tests usually produce vertigo, and hence are difficult of application, but in the normal state the cathode closure produces a slight sound which is distinctly louder than that caused by the anode closure. If the auditory nerve is diseased its reaction is changed. The anode closure sound is greater than the cathode closure sound, and the sound may be heard when the current is broken. In the majority of cases of auditory nerve deafness the vestibular nerve is also affected and vertigo is associated with the deafness.

Deafness from acoustic nerve disease or degeneration occurs occasionally primarily. It may be a congenital condition due to some defective development either in the nerve or in the organ of Corti, under which circumstances the child is never able to hear and never learns to speak. This is the common cause of deaf-mutism. It is an interesting fact that such deaf-mutes are not made giddy by ordinary methods and do not suffer from seasickness. In other cases an atrophy of the acoustic nerve appears to be due to a congenital defect in its power of nutrition, and deafness comes on in early adult life. This is a peculiarly hereditary affection, many members of the same family being subject to the disease. Thus I have known of one large family in which it has appeared through four generations, each generation having had several members who developed the affection. I know another family in which four of seven sons have inherited the deafness of their mother, who had inherited her deafness from her father. They have several uncles and cousins who are deaf. Such cases must be due to a defective vitality in the nerve trunk, and are to be regarded as primary auditory atrophy, the nerve dying before the rest of the body, just as the teeth, hair, and genital organs die before the rest of the body. Syphilis, either hereditary or acquired, may cause acoustic nerve atrophy.

Deafness from acoustic atrophy may develop in the course of locomotor ataxia, of general paresis, and of disseminated sclerosis. I have also seen deafness from auditory atrophy develop in the course of tumors of the brain lying upon the base in such position as to involve the auditory nerve. In syphilis of the base of the brain deafness occasionally develops. Injuries of the head or ear may cause deafness. This is usually attended by vertigo and is due to hemorrhage in the ear. Such hemorrhages may occur spontaneously in the infectious diseases or in states of anæmia. The use of quinine may cause degeneration of the eighth nerve. A functional deafness occurs in hysteria from suspension of activity in fibres of the auditory nerve or in its centres. This form of deafness comes on more suddenly and is more complete than any other type.

The prognosis in deafness from auditory disease is unfavorable, though occasionally counter-irritation over the petrous portion of the temporal bone, and treatment by strychnine, is accompanied by some improvement, but the nerve is not open to any direct treatment, and electrical excitation is of no benefit. Hypodermic injections of pilocarpine, $\frac{1}{4}$ grain, are said to be of some service. Charcot recommended quinine, 15 grains a day, to be kept up some weeks, but the majority of aural surgeons are opposed to this form of treatment.

Tinnitus Aurium. Irritation of the auditory nerve in the organ of Corti, or in the middle ear, or in the course of the nerve, may produce subjective sounds which are appreciated as a ringing or roaring in the ears or head, or a buzzing sound supposed to be outside of the ear. This usually indicates disease of the organ of Corti, either of the nature of disturbances in the circulation or of a beginning auditory atrophy, and if it is followed by progressive deafness the diagnosis of auditory atrophy may usually be made. In at least 80 per cent. of the cases of tinnitus aurium some central deafness can be detected by tests with the Galton whistle or the tuning-fork, even when the patient is not aware that he is deaf. Tinnitus aurium may, however, be due to anæmia. Thus, fainting is frequently preceded by a sound in the ear. Gout is a cause of this symptom. A chronic anæmia with reduction in the number of red corpuscles may cause tinnitus. In these cases the tinnitus is probably due to vibrations of the blood current in the carotid artery, and can be arrested temporarily by pressure upon the carotid artery. Intracranial aneurism causes a sound similar to this. It is because of this blood origin of tinnitus that the symptom develops in states of cachexia and in states of general malnutrition. Direct irritation of the auditory nerve, such as occurs in all forms of otitis media and otitis externa, may produce tinnitus, and anything which interferes with the free circulation of air in the external auditory canal, such as covering the ear with the hand or applying a shell to the ear, will give rise to a perception of unusual sounds. Some drugs, especially quinine and the salicylates, cause tinnitus, and if too long continued, total deafness. In hemorrhagic affections of the ear (Ménière's disease), where the disease lies in the second or vestibular division of the auditory nerve, both tinnitus and deafness are present. A cracking noise due to contraction of the muscles near or about the Eustachian tube is not the same as tinnitus.

The treatment of tinnitus aurium is very unsatisfactory unless the cause, such as anæmia, or gout, or malnutrition, or local disease in the ear, can be removed. But when the tinnitus is the first symptom of an auditory atrophy no cure can be expected. Bromides are of some service, as are also belladonna and cannabis indica.

II. The second division of the auditory nerve is called the vestibular nerve. It is the nerve of equilibrium. It arises from the bipolar cells of the ganglion of Scarpa, whose dendrites come from the ampullæ of the semilunar canals and utricle of the labyrinth. The axones of these cells pass, with the cochlear division of the nerve, to the side of the pons, where they enter directly the lateral portion of the pons (Fig. 204) and terminate about three groups of cells: the cells of Deiters' nucleus, the cells of Bechterew's nucleus, and the cells of the central or posterior nucleus lying upon the floor of the fourth ventricle.

The cells of Deiters and Bechterew's nuclei have numerous connections with other parts of the central nervous system, especially with the cerebellum, with the nuclei of the oculomotor nerves (sixth fourth, and third), with various nuclei in the tegmentum, and with the olivary bodies. Some axones of Deiters' cells pass downward through the formatio reticularis to the motor nuclei of the cervical nerves in the spinal cord, and are probably connected either directly or by means of association fibres with the nuclei of the nerves of the head and back. Its connection with the cerebellum is made by fibres which pass by way of the inferior and middle peduncles of the cerebellum to the flocculus and vermis. Each nucleus is also connected by means of direct and indirect neurones with the cerebellum of the opposite side, which it reaches through the corpus trapezoideus and transverse fibres of the pons. The posterior nucleus of the vestibular nerve is connected by direct tracts through the corpus trapezoideus and fillet and through the formatio reticularis with the upper portion of the brain axis, the corpora quadrigemina, corpora geniculata, and optic thalami. It is also connected with the cerebellum by tracts which pass with those from Deiters' nerve. The existence of a tract to the cortex through the internal capsule has not yet been demonstrated, and no cerebral centre or area controlling equilibrium has been discovered. The weight of evidence in favor of the cerebellum as the chief organ of equilibrium is overwhelming, and it seems needless to suppose that a cerebral area will be discovered. It is evident, therefore, that the semilunar canals in which impulses are received whose object is to make us aware of our position in space and to direct the whole system of movements by which our equilibrium is preserved, have a most widespread connection with the central nervous system and an especially close connection with the cerebellum.

A moment's consideration of the phenomena of equilibrium will convince us of the great importance and complexity of the nervous mechanism controlling it. The body is constantly held in a state of balance, and no movement of any degree can be made in any part without a compensatory movement to preserve the centre of gravity. The slightest variation in our position which would tend to the loss of balance is immediately perceived and immediately corrected. Furthermore, the balance is maintained by an interaction of visual sensations, labyrinthine impressions, and muscular sensations, all of which enter into this function, as can be seen by the disturbance of balance occasioned by double vision, or by labyrinthine disease, or by a loss of the muscular sense, as in locomotor ataxia. While consciousness takes cognizance of this complex matter of equilibrium, the primary centres which receive the impressions and regulate the activity that they arouse are located in the cerebellum. This is the great organ of equilibrium, and it therefore receives impressions directly from the eyes, from the semilunar canals, and from the muscles of the limbs. The most important impulses in the preservation of equilibrium, however, are derived from the semilunar canals through the vestibular nerve, and affections of this nerve are attended by very marked and very intense symptoms of vertigo. Vertigo may be due, as is usually the case, to affections of the terminal organs in the semilunar canals, or to affections of the vestibular portion of the auditory nerve in its course to the cerebellum, or to lesions of the pons, or to disease of the cerebellum. Disease of the crura not involving the corpora quadrigemina, or of the superior peduncles of the cerebellum, through which the ocular impulses reach the cerebellum, also produces vertigo.

Vertigo is a symptom of uncertainty of position, attended by a sensation as if the body itself were being revolved in any one of all possible directions, or as if objects outside of the body were turning rapidly. It causes balancing movements, oscillation of the eyeballs, and staggering in the gait. It is attended by great uncertainty and distress of mind, the patient grasping at adjacent objects for support as he feels himself falling, or throwing himself flat upon the floor and clinging to anything in the vicinity for support. A distinction is sometimes made clinically between the sensation of objects outside of one's self turning, called objective vertigo, or of one's body being in a state of revolution, called subjective vertigo. But this distinction of symptoms cannot be referred to any difference in the lesion in any part of the mechanism. Vertigo may be produced by a rapid revolution of the body or by the passage of a galvanic current through the head. It is thought that hemorrhages in or destruction of the superior vertical or sagittal semilunar canal (anterior, of Retzius) produce a sense of revolution in the entire body, as if the head were going down and the feet up, the head going toward the side of the lesion. It is thought that hemorrhages in the inferior or horizontal semilunar canal (external, of Retzius) produces a sensation of rotation of the body around its vertical axis toward the side affected. It is thought that a hemorrhage in the posterior vertical or frontal semilunar canal (posterior, of Retzius) produces a sensation of falling toward the side of the lesion. But, inasmuch as these three canals open into one another, further investigation is necessary in order to establish these assertions.

Vertigo is usually associated with intense vomiting and great mental distress. A rapid action of the heart and general relaxation of the bloodvessels, causing profuse sweating and a feeling of great faintness, commonly attend vertigo. It seems probable that anything which causes an intense irritation of the nuclei of the vestibular nerve produces a sympathetic disturbance of the pneumogastric centre which lies adjacent to it. Conversely, we know that irritation coming from the pneumogastric nerve to the brain, such as occurs in gastric disorders or in heart disease, may produce a secondary vertigo.

Gastric vertigo is easily differentiated from vertigo due to vestibular disease by the absence of tinnitus and deafness or pain in the ear. Vertigo due to cerebellar affection is also rarely followed by deafness or by pain in the ear.

Vertigo is such a common symptom that it is well to classify its causes in order to reach a diagnosis.

I. Vertigo from affections of the auditory apparatus.

1. Vertigo from disease in the outer and middle ear. This form is moderate, is attended by some staggering or inability to stand, and by roaring in the ear and deafness. Any disease of the ear may cause this type of vertigo in greater or less degree. Thus a plug of wax or any irritation in the ear from sea water, picking the ear, or boils in the auditory canal may produce giddiness. Such affections are visible. Any form of otitis media is attended by vertigo, which may be one of the most distressing symptoms. It is attended by pain in the ear, by changes in the appearance of the drum membrane, and eventually by a discharge of pus. It is somewhat relieved by inflation.

Treatment should be directed to the ear disease.

2. Vertigo from disease of the inner ear—*Ménière's disease.* This form is very intense and distressing, begins with a loud report in the ear, is attended by prostration, inability to stand and pallor, coldness and sweating, and vomiting, and is always associated with tinnitus and deafness. There may be nystagmus, great mental distress, and a loss of consciousness. It may come on suddenly, and then constitutes the symptom-complex named after Ménière, who first described it. Ménière's disease is due to hemorrhage in the semilunar canals, and comes like an apoplectic stroke, but is not accompanied or followed by paralysis. It may come upon a person in perfect health, or it may be preceded by ear disease or by arterio-sclerosis. If the attack is recovered from it is usually followed by permanent deafness and by other attacks, and any exertion or mental excitement may bring on an attack. The patients dread such attacks and live in terror of them. The attacks, at first lasting a few minutes only, become more and more frequent and severe, as a rule, and finally the patient suffers from constant dizziness, becomes extremely nervous and apprehensive, and dies in an attack. Occasionally the first attack is not followed by recurrence and all symptoms excepting deafness pass off.

The treatment of vestibular vertigo as a symptom is by rest in bed, applications of ice-packs to the ear or blisters behind the ear, and the free use of bromide, chloral, and nerve sedatives, or, in case these fail to relieve, by hypodermic injections of morphine.

Voltolini has described an affection which he considers due to primary inflammation of the labyrinth. This is known by his name. The disease occurs in children, but adults are not exempt. It resembles acute meningitis. It begins suddenly with high fever, general cerebral symptoms, of which vertigo and vomiting are the most prominent, and consciousness soon becomes obscured. After a few days these symptoms subside, but the child still suffers from dizziness, staggers in walking, and is found to be deaf. The staggering gradually passes off, but some degree of deafness remains. The lesions found have been plastic exudations with destructive processes in the semilunar canals. It is practically impossible to differentiate Ménière's disease from Voltolini's disease in the early stage, and even in the later stage it is very difficult. In Ménière's disease the attacks of vertigo recur. The treatment should be by counter-irritation behind the ears, hot baths, antifebrile remedies, purgatives, and bromides in large doses.

3. Vertigo from disease in the vestibular nerve is attended by symptoms quite like those in Ménière's disease, but is often accompanied by forced movements or by staggering or falling in some definite direction.

Thus in a case of a fireman seen at the New York Hospital, who by a fall had fractured the base of his skull and torn the left auditory nerve, the most marked symptoms during the three days preceding his death were an absolute deafness in the left ear, a constant agonizing sensation of rotation of the body in its longitudinal axis, and consequently a constant turning of the body in the opposite direction in the endeavor to correct the subjective sensation. Unless firmly held this man constantly revolved upon his bed, and when held complained of the most intense distress. In this case the vertigo was attended by extreme exhaustion, vomiting, utter prostration, rapid pulse, and death from heart failure.

Raymond[1] in his lectures has described similar cases.

4. Vertigo is a frequent symptom in disease of the pons Varolii. Tumors upon the base of the brain, syphilitic exudations or vascular lesions in the pons, and abscess or tumors in the cerebellum compressing the pons irritate or destroy the vestibular nerve or its nuclei. In these cases the vertigo is usually mild in degree. It is accompanied by a staggering gait, the tendency being to stagger to one side, in the majority of cases, but not invariably, toward the side of the lesion. The existence of other general symptoms of tumor, abscess, syphilis of the brain, sclerosis, or vascular lesions, and the presence of other cranial nerve symptoms enable a diagnosis to be reached. Such a tumor is shown in Fig. 232, page 588.

II. Vertigo from affections of the ocular apparatus.

Any sudden attack of diplopia due to paralysis of an ocular muscle is liable to be attended by vertigo. Thus lesions of the third or sixth nerve or ophthalmoplegia externa produce giddiness and cause uncertainty of gait. This form of vertigo is often attended by nystagmus. It ceases when the eyes are closed and when the patient does not use the affected eye.

III. Vertigo from affections of the muscular sense.

While staggering gait is a frequent symptom in locomotor ataxia, vertigo is less constant. It occurs, however, with such frequency as to require mention. Charcot pointed out that it develops in those cases whose general sensibility and muscular sense are profoundly affected. It may be due in some ataxics to a primary atrophy of the vestibular nerve.

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[1] Clinique des Maladies du Système Nerveux, 1900, IVieme série.

IV. In the three types of vertigo thus far studied it is evident that the lesion interferes with the conduction to the cerebellum of those impulses which are necessary to an appreciation of one's position in space. Lesions in the cerebellum itself, the organ of equilibrium, are almost always attended by vertigo. This is particularly true of lesions of the vermis or central lobe and by lesions of the flocculus, the small polyp-like lobes on the base. It is thought that lesions of the vermis in its anterior part cause staggering forward, and in its posterior part, backward. Such staggering is due to a sensation of falling in the opposite direction. Any form of lesion may cause this symptom, but it is particularly constant in tumors of the cerebellum. The diagnosis can be reached only by observing the general signs of cerebellar disease in addition to vertigo. In congenital defective development of the cerebellum, vertigo, nystagmus, and staggering appear early. In Marie's disease they are permanent symptoms.

Neurasthenia and hysteria often produce vertigo of a mild and transient variety, which is probably cerebellar. The so-called "essential vertigo," consisting of attacks of vertigo with no other symptoms, is probably a functional neurosis of the cerebellum, and occurs in attacks quite like epileptic attacks, and is usually to be treated as a form of epilepsy. Epileptic attacks are almost uniformly preceded or attended by vertigo.

Gertier, of Geneva, has described a peculiar disease, which has been seen in Switzerland and France, and is known as Gertier's disease or *vertige paralysant.* It also occurs in Japan, where it is known as *kubisagari.* It is characterized by a series of sudden, short attacks of vertigo, with ptosis and somnolence, pain in the neck, with paralysis of the neck muscles, allowing the head to fall forward, or general weakness or even general paralysis, with thickness of speech but no loss of consciousness. The attacks last a few minutes, recur every few minutes for several hours, leaving the patient fatigued but well in the interval. They increase in frequency during the hot weather, but cease in winter. The disease lasts from one to five months. In the intervals the patients appear to be in good health. Gertier believes that exhalations of marshes and stables have something to do with its occurrence. He also finds it more common in those who work in a bowed position, and in those who are much fatigued and are neurotic and emotional. It is observed in laborers on farms. It has been seen in epidemics about Geneva, but no fatal cases have been observed. In the cases in Japan, Miura found an exaggeration of the knee-jerks. Dejerine thinks that the peculiar fits to which cats are liable are of this same nature.

V. Vertigo may occur as a general symptom in disease of any kind within the cranium. It is especially frequent in diseases causing a sudden change in intracranial pressure. Thus the effort of straining at stool, lifting a heavy weight, or running fast may cause vertigo.

Apoplexy is usually preceded or attended by vertigo, and brain tumors or abscesses, sclerosis or syphilis, no matter what their location, frequently cause vertigo. It is probable that in all these conditions some irritation is set up in the cerebellum or some defective circulatory condition is produced in it sufficient to disturb its function. This form of vertigo is not very intense and never causes forced movements, nor is it usually sufficient to cause unsteadiness of gait. It is often attended by vomiting and sometimes by convulsions. It is not usually associated with deafness. It is not relieved by lying down. It is worse on waking after sleep. It may be relieved by hot baths.

VI. Vertigo may also be due to irritation in the cerebral centres produced by conditions of the blood. Thus certain toxic agents, alcohol, nicotine, coffee, opium, quinine, the salicylates, the coal-tar products, and many other drugs, cause vertigo. Vertigo is an early symptom in auto-intoxication from stomach or intestinal fermentation and in the early stage of almost all infectious diseases; gout, uræmia, and diabetes may produce vertigo.

VII. Vertigo may be due to anæmia of the brain from heart failure, chronic valvular disease, particularly aortic obstruction, and mitral regurgitation, and from arterio-sclerosis. It attends a fainting fit and severe hemorrhage. It is also produced by venous congestion of the brain, as after violent exertion, straining at stool, or lifting heavy weights.

VIII. There are some forms of vertigo which are reflex in origin. These are so-called stomach vertigo and laryngeal vertigo. The nuclei of the vagus nerve lie close to that of the vestibular nerve. Any irritation reaching one may easily extend to the other or be conveyed to the other by association fibres; hence vertigo and vomiting are commonly associated symptoms. Stomach vertigo is a form of vertigo occurring in sudden attacks associated with belching of gas, or raising of acid fluid, with pain in the stomach and with various symptoms of dyspepsia, and relieved by vomiting. It may attend any ingestion of food or it may follow a meal after two or three hours. It is not infrequently a symptom in dilatation of the stomach, but rarely in organic disease such as ulcer or cancer. It is more frequent in patients who are anæmic, poorly nourished, or neurasthenic. It not uncommonly develops in old persons. It usually yields promptly to treatment directed to the stomach. Large doses of bicarbonate of soda, bismuth, or salol relieve it at once, especially if taken in hot water; and care in the diet, with proper treatment for indigestion, prevents a recurrence of attacks.

Laryngeal vertigo is a symptom occurring usually in the course of tabes, especially when other laryngeal symptoms have been present. It occurs in a sudden attack, with cough, sense of suffocation, dyspnœa, and feeling of strangling. It lasts only a few moments, but may recur several times. It may be so intense as to cause asphyxia and loss of consciousness, and it has been fatal in some cases.

Nasal vertigo may also be mentioned, though the reflex irritation here arises in the fifth nerve. It occurs in cases of polyps and obstructive mucous thickening in the nose, and is relieved by the removal of the original disease.

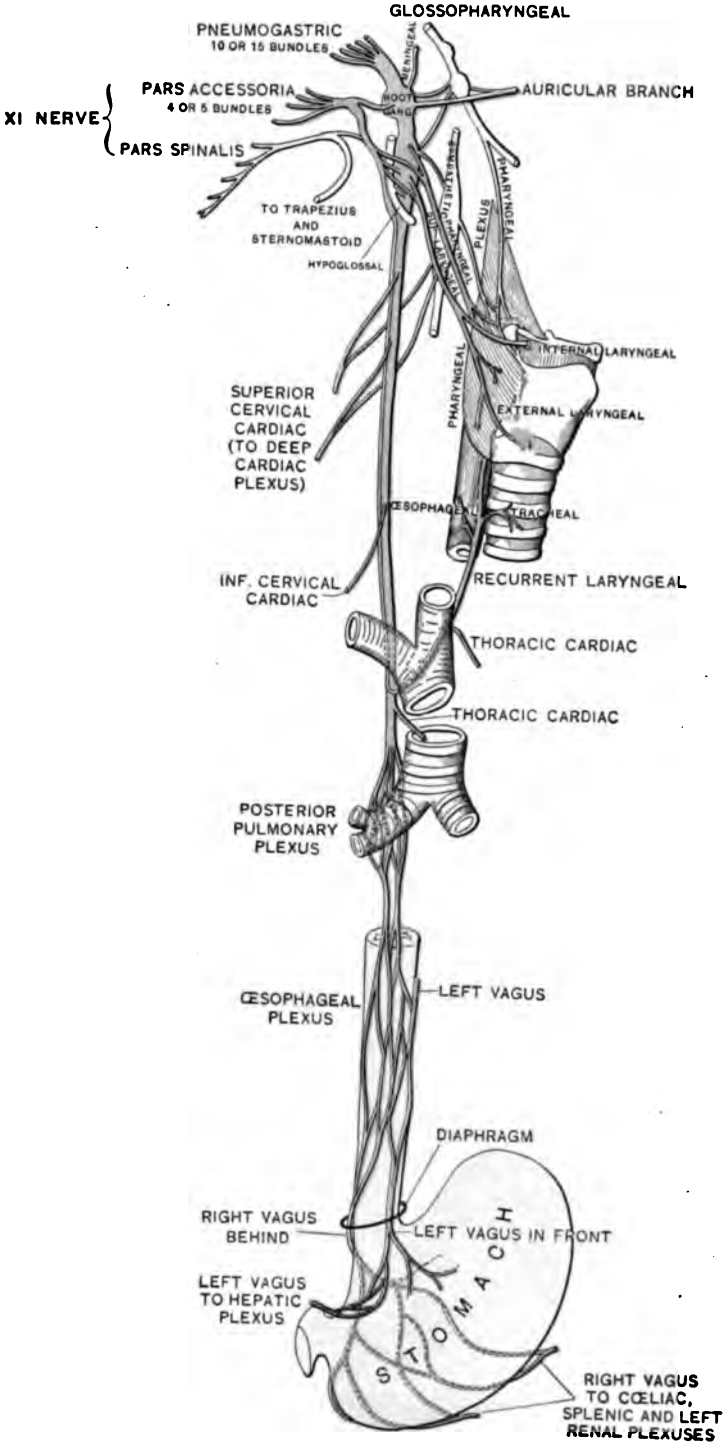
Other diseases supposed to excite vertigo by reflex irritation are uterine and ovarian affections.

IX. Psychical disturbances sometimes produce vertigo. The dizziness one feels when on a height is an example; and as any mental experience may be revived in memory with an intensity equal to its perception, hallucinatory vertigo is a common symptom. It occurs in many cases of neurasthenia and hysteria, in hypochondria, and in traumatic neuroses. This form of vertigo is rarely attended by staggering; it belongs to a class of symptoms known as phobias or morbid fears, the fear of falling being often attended by vertigo. In such cases suggestion is of great use in treatment. Thus in one case so severe as to keep the patient in bed a cure was produced by convincing her that if her head was held steady she would feel no dizziness. The head was held at first by the hands, later by a firm head splint attached to the back, and after two or three days the symptom disappeared.

THE NINTH NERVE: THE GLOSSOPHARYNGEAL NERVE.

This is a mixed nerve containing both sensory and motor fibres. Its sensory branches come from the tongue, larynx, and respiratory organs and middle ear. It is the nerve of common sensibility of the pharynx and palate and of the middle ear. It is thought that it conveys the sensations of taste from the posterior third of the tongue, those from the anterior two-thirds passing in through the lingual nerve. But as division of the root of the trigeminal nerve has caused a total loss of taste in some patients, there is some doubt as to this function. (See page 659.) The ganglia of origin are the ganglion jugulare on the glossopharyngeal nerve and the two ganglia on the vagus nerve. (Plate XXVI.) The sensory fibres enter the side of the medulla just outside of the upper part of the olivary body, and terminate partly in the gray matter on the floor of the fourth ventricle and partly in a thin column of gray matter like the substantia gelatinosa, lying along the inside of the solitary bundle in the formatio reticularis. The sensory fibres on entering the medulla form a part of this bundle with other sensory fibres of the vagus nerve and turn downward, passing as low as the upper cervical segment of the spinal cord. The solitary bundle is therefore similar in its structure to the descending root of the fifth nerve. It has also been called the respiratory bundle of Krause, as its division suspends respiratory motions. Its fibres end in tufts which lie in the substantia gelatinosa which borders its inner surface. From the cells of this substance new fibres arise which enter the formatio reticularis and the lemniscus, and so ascend to the brain; but their termination

PLATE XXVI.



Distribution of Right Pneumogastric Nerve. (W. Keiller.)

in the cortex has not yet been ascertained. (See Fig. 181, *5*; page 445.)

The motor branches of the glossopharyngeal nerve arise from a column of cells known as the nucleus ambiguus, which lies in the lateral part of the formatio reticularis. (Fig. 181, *5*.) They curve upward, inward, and backward, then outward, forming a knee like that of the facial nerve. They issue from the side of the medulla posterior to the olivary body and pass out of the skull through the jugular foramen. They go to the muscles of the larynx, œsophagus, and pharynx, and preside over the functions of respiration, swallowing, and phonation. (See also Fig. 198, page 475.)

The origins of both the motor and sensory fibres of the glossopharyngeal nerve are so intimately mingled with those of the vagus nerve that separation between them is impossible. The following table of Spencer shows the probable functions of these two nerves:

THE DISTRIBUTION OF THE NINTH, TENTH, AND ELEVENTH NERVES.

	Afferent.	*Efferent.*
Upper roots, ninth nerve.	Respiratory regulating fibres; respiratory exciting fibres; (inspiration) inferior laryngeal.	Cricothyroid, stylopharyngeal, œsophagus, pharyngeal constrictors.
Middle roots, tenth nerve.	Respiratory inhibitory fibres; (expiration) bronchial.	Gastric branches, bronchial muscles.
Lower roots, tenth nerve, accessory eleventh nerve.	None.	Inferior laryngeal, cardiac inhibitory, levator palati.
Spinal accessory eleventh nerve, spinal part.	None.	Sternomastoid, trapezius.

THE TENTH NERVE: THE VAGUS OR PNEUMOGASTRIC NERVE.

This, like the ninth nerve, contains both sensory and motor fibres. Its sensory branches come from two ganglia on its trunk, of which the upper ganglion is large, oval, and resembles the posterior spinal ganglia, and the other is long and irregular, the cells being scattered among the fibres of the nerve. (Plate XXVI.) These sensory fibres enter the side of the medulla outside of the olivary bodies and pass in several directions. Some go inward and backward to terminate in a mass of gray matter which lies on the floor of the ventricle laterad of the twelfth nerve nucleus in the medulla and dorsad of it in the upper cord, a mass which projects upon the floor of the fourth ventricle forming the ala cinerea. This is the respiratory and cardiac centre. Others join the ninth nerve fibres and enter the solitary bundle, descending in it and ending in the substantia gelatinosa which borders it on its inner side. From these nuclei new fibres arise which enter the formatio reticularis and ascend to the brain, but their course and termination is yet undetermined.

The motor branches of the vagus arise from the cells of the nucleus ambiguus, turn inward, curve about in the formatio reticularis, and then turn outward and make their exit on the side of the medulla, where they form the great trunk of the vagus nerve.

The destination of some of these sensory and motor fibres has already been shown in the table, page 683.

There are also fibres which pass to the meningeal branches of the fifth, to the auricular branches of the seventh, and to the carotid and abdominal plexuses of the sympathetic system.

While it might be supposed from the anatomical distribution that a lesion of the glossopharyngeal nerve would cause anæsthesia and paralysis of the pharynx and larynx and difficulty in swallowing, there are no cases on record of lesion of this nerve trunk.

In bulbar paralysis the nuclei of the nerve are affected, and paralysis of the œsophagus and of the pharynx and larynx are the result; but here, too, it is not possible to distinguish sharply between symptoms due to lesion of the ninth and tenth nerves.

The great length of the vagus nerve exposes it to many injuries and to compression by tumors at any point in its course. Acute neuritis of the vagus from cold or rheumatism is not known to occur. Certain poisons, especially that of diphtheria, and alcohol and certain drugs, atropine, veratrum, aconite, and digitalis, etc., appear to have a special selective action upon the nerve and may produce symptoms referable to it. While the distribution of the nerve to the heart, lungs, and viscera is a very wide one, we know but little regarding the symptoms actually produced by its lesion. The nerve is a bilateral one in its action, and in many cases where it has been divided by stab wounds, or in the course of surgical operations upon the neck, very few symptoms have ensued, though experiments upon animals would indicate that a division of the left nerve should be attended by a very rapid heart action and by a slowing of respiration. This has occasionally occurred when surgeons have included the vagus in tying the left carotid artery. The branches of the nerve which pass to the larynx and supply all the muscles of the larynx are most important, and their injury gives rise uniformly to very marked symptoms. These symptoms have been carefully studied by Gowers, whose table is so complete that it is here reproduced.

Symptoms.	*Signs.*	*Lesions.*
No voice; no cough; stridor on deep inspiration only.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low-pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless; the other normal or even beyond the middle line in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
No voice, perfect cough; no stridor or dyspnœa.	Cords normal in position, and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

It will be remembered that the recurrent laryngeal nerve in its long course around the aorta is frequently compressed by aneurisms, and the result of such compression is usually paralysis of the vocal cord on one side, with whispering voice and difficulty of respiration. The abductors of the glottis being paralyzed, the opening of the glottis is not sufficient during the act of respiration, and hence a whistling sound or wheeze and a sense of suffocation results. In a few cases of injury of the vagus in this part of the neck some anæsthesia of the throat back of the palate has been observed, together with disturbance in the act of swallowing. The reflex act of swallowing from irritation of the pharynx is also suspended in these cases.

An occasional paralysis of the vagus nerve occurs as a complication of locomotor ataxia, and spasm of the larynx with suffocation may also occur in this disease.

The diagnosis of vagus paralysis can be made when the recurrent branches of the nerve are affected and disturbances in respiration and in the voice develop, or when there is a distinct anæsthesia of the pharynx, with difficulty of swallowing, not due to paralysis of the palate or to paralysis of the tongue. Rapidity of the heart is not a sufficient sign to warrant a diagnosis, and there are no lung, gastric, or visceral symptoms that are characteristic of the disease, the majority of the statements made in the books being purely theoretical and not based upon clinical facts or pathological findings.

In the course of bulbar paralysis the nuclei of the ninth and tenth nerves become involved and undergo degeneration. This is indicated clinically by the development of respiratory and laryngeal palsy and by a very rapid heart action. These may lead to a fatal result. The occurrence of hemorrhage or of embolism in the medulla involving the vagus nuclei is the cause of sudden death in some cases of apoplexy. The vagus nucleus when disturbed in its function can no longer secure normal breathing or heart action, hence Cheyne-Stokes respiration and intermittent or very rapid heart action are supposed to be symptoms referable to its functional disturbance. Vomiting is also supposed to be due to its irritation. This form of vomiting is sudden, explosive, not preceded by nausea and is very violent, and is attended by great prostration. It occurs occasionally in tumors or abscess of the brain in any location and also in meningitis of the base.

The vagus nerve is often involved in the course of multiple neuritis due to any of the numerous causes which produce that disease. The signs of its implication are rapid pulse, general vasomotor paralysis, shown by cyanosis, œdema, profuse sweating, and respiratory paralysis, causing irregular, labored breathing. Under these conditions, which are of a very unfavorable prognostic importance, stimulation by alcohol, strychnine, and digitalis, and by nitroglycerin are imperative. (See page 112.)

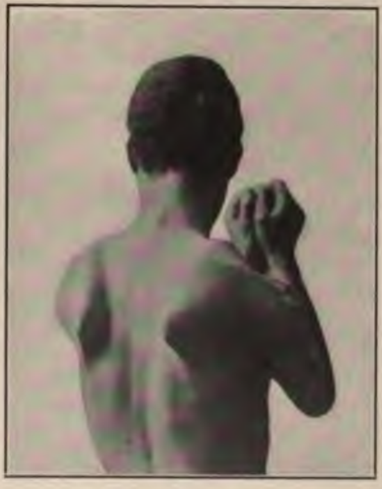
Spasm of the œsophagus is not to be considered as an evidence of affection of the ninth nerve, but is a functional nervous disease usually hysterical in origin or due to organic disease of the œsophagus.

THE ELEVENTH NERVE: THE SPINAL ACCESSORY NERVE.

Paralysis of the Accessorius. The eleventh nerve arises from a long column of cells which lie in the lateral part of the anterior horn in the upper five cervical segments of the spinal cord. The roots issue from the lateral surface of the cord and ascend to the brain, where they receive further fibres from the lateral portion of the medulla, the accessory portion, which really is independent of the nerve and belongs to the vagus, and then make their exit from the skull through the jugular foramen. It is rarely injured by disease within the spinal canal or skull, though fractures and injuries may affect it. The usual cause of paralysis of the spinal accessory is cold, injury, or pressure by tumors, swollen glands, or abscess in the neck. I have seen the condition in a young girl due to accidental division of the nerve when deep-lying glands were removed from the neck.

Symptoms. The symptoms of paralysis of the spinal accessory nerve are paralysis of the sterno-cleido-mastoid muscle and of the trapezius muscle. The sterno-cleido-mastoid turns the head and elevates the chin, so that when the right muscle contracts the patient looks over his left shoulder. The trapezius turns the head backward and over toward the shoulder and also elevates the shoulder. Other muscles act in association with these two muscles, and hence a lesion of the spinal accessory nerve causing a paralysis of these muscles is not apparent until the patient is examined; then it is found that the motions which they accomplish are not done with the usual amount of power, and a slight amount of resistance renders the patient incapable of turning the head or of elevating the shoulder. The greatest defect lies in the elevation of the arm above the horizontal line; the trapezius counteracts the action of the serratus, and if it is paralyzed the serratus rotates the scapula in such a manner as to produce a slight elevation upon the side of the neck by the projection upward

FIG. 266.



Paralysis of the right spinal accessory nerve. The inner angle of the scapula projects upward on the neck and the shoulder-blade projects unduly.

of its inner angle. Fig. 266 demonstrates this action, and shows that under these circumstances the mechanism of movement in the shoulder will be seriously hampered. When the patient brings his arm forward and outward the edge of the shoulder-blade is no longer held tightly to the chest, and hence projects upon the back. Fig. 266 shows this deformity.

The diagnosis is made by the discovery of the weakness of the muscles, of the imperfect elevation of the arm, of the deformity, and of the existence of reaction of degeneration to the electrical examination.

The prognosis will depend upon the possibility of removal of the cause, and the treatment is by massage and electricity. In cases which do not recover it may be possible by appropriate apparatus to produce a fixation of the shoulder-blade in its proper position, and thus aid in the movement of the arm.

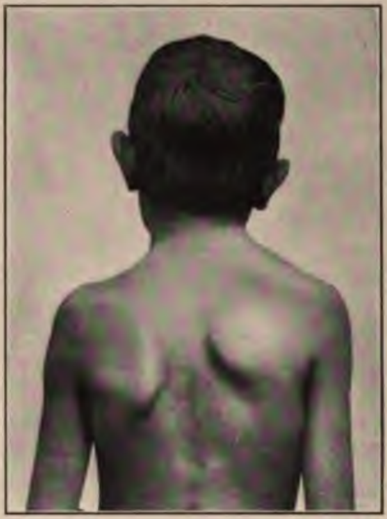
Torticollis: Wryneck. Congenital lesions of the eleventh nerve due to defects of development or injuries at the time of birth sometimes result in a defective development of the sterno-cleido-mastoid muscle, which fails to grow in proportion to the other muscles, and hence becomes permanently shortened. In these cases as the child grows the head is slowly turned away from the affected side, the chin approaching the shoulder, and it cannot be turned straight or to the opposite side on account of the permanent shortening of the affected muscle. In such a condition there is no spasmodic contraction of the muscle and no twitching of the head, but the lesion is a wryneck fixed and permanent. This condition is easily remedied by a division of the tendons of the sternomastoid muscle, when the other muscles secure a proper position and proper movement of the head.

Spasmodic torticollis is a spasmodic contraction of any or all of the muscles moving the head upon the trunk, but is considered here because of the frequency with which the muscles supplied by the eleventh nerve are affected. It consists of a quick, sharp contraction of the sternomastoid or of the trapezius muscles, causing a sharp, quick turning of the head toward one side, as if the patient were looking over his shoulder. In some cases this contraction is so sudden as to cause an almost lightning-like twitch of the head. In other cases the contraction is slower when the head is turned slowly, but none the less firmly to the side. Twitching occurs at intervals, may take but a second, and be succeeded by many other twitchings, so that the head turns eight or ten times in a minute, or it may be much slower, and then the head is frequently held for a minute or more in a fixed position. As soon as a relaxation of the muscle occurs the patient voluntarily turns the head back, only to find it jerked about again with much force and persistency. As time goes on in this affection the twitchings become more severe and either more rapid or more prolonged in duration. The latter is very common, so that these patients after a time find the head permanently fixed in the abnormal position, though the occasional relaxation of the spasm allows them to turn the head back occasionally. The usual form of torticollis is the one in which the sternomastoid muscle alone, or the upper fibres of the trapezius alone, or these two muscles together are affected. If the sternomastoid alone is involved the chin is elevated and drawn toward the opposite shoulder. If the trapezius alone is involved the chin is elevated to a greater degree and the head is thrown backward. If both muscles act together the entire head is turned over toward the affected side and rotated with the chin toward the opposite shoulder. The affection is liable to be of long duration, the spasm continuing for many weeks, with varying degrees of intensity. It usually relaxes during sleep. It may be produced by any voluntary effort, especially the acts of reading or looking fixedly at any object which involves the voluntary fixation of the head or eyes. Patients usually find that there is some point upon the neck or head, pressure upon which will temporarily arrest the spasm, and they are inclined to produce pressure upon this point either by resting the head upon the back of the chair or lying with the support of a pillow or compressing this point by the hand. For one patient who had a serious form of spasmodic torticollis which failed to respond to any treatment I devised a mechanical apparatus containing a lever which pushed a pad against the lower part of the occiput upon the left side, which pad could be made to press more firmly by a movement of adduction of his arm, and this apparatus was worn for many months with great relief. The spasm is not, as a rule, accompanied by any pain or by any sensory disturbances, though the muscles may become wearied by their constant contraction and may feel tired and stiff. Occasionally there is acute neuralgia in the back of the neck or in the occipital nerve. The act of walking or talking may intensify the spasm, and any attempt at effort of the arms occasionally is attended by an increase in the contractions. Thus one patient at present under my observation is prevented from writing by the occurrence of the spasm. The muscles that are affected may be seen and felt to contract, but show no changes to the electrical reaction.

The spasm may extend from the sternomastoid and trapezius muscles to any or all of the deeper muscles moving the head. The splenius is affected in many cases, and in a few cases the deeper muscles of the back of the neck are all involved in the spasm. Under these circumstances it is not unusual for the spasm to extend to the opposite side or to affect the superficial muscles on one side and the deeper muscles on the other, which act conjointly in turning the head. Symmetrical muscles are rarely affected. If they are they usually act in harmony, producing a throwing of the head forward upon the chest or a turning of it backward and an elevation of the chin. Sometimes in serious cases of long duration the spasm extends to the muscles moving the shoulder, the rhomboids, the levator anguli scapulæ, or the serratus or pectorals; and in these cases movements of the arm are frequently associated with those of the head. (See Fig. 267.) The arm may be adducted and flexed, wrist and fingers flexed, or it may be thrown backward behind the body. Occasionally these spasms become so intense that the muscles of the back are also implicated, and the patient is no longer able to walk in an upright position and even while lying down is drawn around to one side, the curve of the spine being convex toward the unaffected side. This I have seen in one chronic case which lasted for five years and resisted all forms of treatment including division of the eleventh nerve. In cases where the head is thrown backward by contraction of the trapezii there is an associated movement of the frontalis muscle, and then the patient not only elevates the chin but has the appearance of looking upward forcibly, the eyebrows being raised. As time goes on the muscles that are affected increase in size on account of their constant exercise, so that the neck becomes perceptibly larger.

The course of the disease is a chronic one. The spasm begins in slight degree, increases in intensity, in frequency of attack and in duration of attack, so that within three months of the onset the patient's life becomes a burden and all occupation has to be abandoned. In a few cases after a period of two or three months the spasms gradually diminish in frequency and cease, but they are particularly liable to recur from time to time, and these patients suffer from attacks of spasmodic torticollis throughout their lives. The disease occurs after any illness. In many cases it continues for over a year with little intermission, sometimes being moderate in degree, at other times increasing in intensity. Even the more severe cases occasionally recover, the spasms gradually subsiding and finally disappearing. It is not attended by convulsions or by cortical epilepsy, though I have occasionally seen it accompanied by considerable mental agitation, a distinctly hysterical mental condition developing as a result of the irritation of the continuous spasm. Cases recorded by Brodie and Gowers in which the spasm ceased during an attack of insanity are interesting.

FIG. 267.



Tonic spasm of right trapezius, rhomboid and levator anguli scapulæ, of two years' duration, following whooping-cough.

There are no pathological observations to determine the exact cause of torticollis. The disease is not primarily muscular nor is it to be ascribed to any lesion of the eleventh nerve or motor nerves to the muscles, inasmuch as irritation of the motor nerves does not cause spasm. It may be regarded as reflex in some cases, like facial tic and blepharospasm, as the result of a sensory irritation in some one of the sensory nerves of the neck. In these cases neuralgia may precede the attack. In the vast majority of the cases no cause can be ascertained. The hypothesis of a cortical origin of this spasm receives a certain amount of support from the fact that the movements are quite similar to those produced by volitional effort, and in many cases muscles which lie upon opposite sides of the body, and are supplied by various nerves, join to produce the movement. The fact that the eyes are not uncommonly turned involuntarily to the side toward which the face looks and that the eyebrows are elevated when the head is turned upward supports this hypothesis. When the arm is involved the motions produced are such as would be produced in a normal individual straining to move the head in an extreme position of rotation. Anyone can substantiate this fact who makes extreme voluntary efforts to turn the head from side to side.

Etiology. The disease is more common in women than in men. It occurs in adult life between thirty and fifty years, though no age is entirely exempt. It usually occurs in persons predisposed to nervous disease by inheritance or subject to other forms of nervous affection, such as neurasthenia, migraine, or psychosis. It is not analogous to chorea nor does it follow attacks of chorea or facial tic. Occasionally it has been known to develop after a fall or an injury in which the head was forcibly twisted. It has been known to occur after exposure to cold and after an attack of rheumatism in the muscles of the neck. It has been known to follow otitis media, and in very many cases some defect of vision has been discovered which indicates that eyestrain may be a factor. It has been ascribed to insufficiency of the ocular muscles and also to myopia and hypermetropia. Both these conditions undoubtedly cause an involuntary straining of the muscles of the neck in the act of vision, and occipital headache. None of these causes, however, seems to me of sufficient moment to originate the disease. I am disposed to regard it as almost entirely a cortical affection dependent upon some irritating cause acting upon the cortical centres which move the head. This irritating cause may be exhaustion from voluntary overaction of the muscles consequent upon defects of vision, and it may be considered as an affection analogous to the many forms of occupation neuroses. If this theory is accepted the functional disturbance is to be located in the entire nervous mechanism involved in the movement of the head, the sensory elements and the motor elements being equally involved and thrown into a state of hypersensitiveness which results in slight sensations producing undue movements.

Prognosis. The prognosis in wryneck must be given with great caution, as the history of various cases shows such varied course. The more sudden the onset, the more intense the spasm, the more widespread the distribution, and the longer its duration the worse the prognosis. The younger the patient, the less severe the affection, the greater the intervals between attacks, and the less the discomfort appreciated the more likely is recovery.

Treatment. The treatment of torticollis is unsatisfactory because we are ignorant of the cause of the disease. Various forms of nerve sedatives have been used. I have found but little relief from large doses of bromide or from bromide and chloral. Valerianate of zinc and asafœtida have given somewhat better results, but the best effects have been obtained from large doses of tincture of belladonna, which may be gradually increased up to full physiological effect and usually cause a distinct improvement in the condition. Cannabis indica has also been of some service in the treatment of this affection. When the spasm is very severe and causes great distress opium in some form may be used, in the extreme cases by hypodermic injections of morphine. This usually gives considerable relief, but unfortunately leads to the morphine habit, to which several of my patients have succumbed. Hypodermic injections of atropine, $\frac{1}{200}$ grain increased slowly up to $\frac{1}{20}$ grain once daily, into the muscles affected have given relief in some cases, but have failed in others. Electricity has no effect whatever. If it is tried it should be in the form of a mild, continuous galvanic current, as it is futile to produce contractions in muscles already in a state of spasm. Attempts to strengthen the opposing muscles by electrical exercise should not be made, for even the greatest voluntary contraction in the healthy muscles is incapable of overcoming the involuntary spasm. Counter-irritation to the back of the neck by means of blisters and by the actual cautery not uncommonly gives a certain amount of temporary relief and may be used frequently if the skin be not injured. Mechanical supports of all sorts are involuntarily sought for by the patient, and apparatus may be devised for producing a constant support of the head, a rod carrying a pad being attached to a corset and thus held in position.

Torticollis has been treated by surgeons by division of the tendons of the muscles. This affords temporary relief, but does not stop the spasm, which returns as soon as the tendons have united. It has also been treated by division or by cutting out a portion of the eleventh nerve where it enters the sternomastoid muscle, or deep in the neck just at its exit from the skull. These measures certainly stop the spasm in the muscles supplied by the nerve divided, but very often after the division of the nerve the motion continues, and then it is discovered that the muscles lying deep in the neck upon the same side or upon the opposite side have originally been affected by the spasm. Keen has divided the upper four cervical nerves in efforts to paralyze these deeper muscles, an operation involving considerable difficulty on account of hemorrhage and on account of the depth to which the wound must be carried. In some cases this has given relief, but after the nerves have regenerated the spasm has returned. In a discussion before the American Neurological Association in 1897[1] the consensus of opinion was against surgical treatment, yet all agreed that it afforded the only prospect of temporary relief in severe cases.

Nodding Spasm. In some children soon after birth or within the first six months of life certain oscillatory motions of the head develop, usually lateral, sometimes vertical, and occasionally a mixture of both. These are very commonly associated with a condition of rotary or lateral nystagmus which appears when any attempt is made to turn either head or eyes. These nodding movements do not seem to incommode the child and are certainly not attended by pain. They are not unlike the nodding movements and slight rotary movements occurring in very old persons, though these are not associated with nystagmus. Nodding spasm is more common in children of neurotic inheritance and children who are badly nourished and the subject of rickets. It develops not infrequently in cases of hydrocephalus in mild degree in the early stage. It also occurs in tumors of the brain in children. The origin of the condition is wholly unknown, though in several cases when children subject to nodding spasm have grown up and have outgrown the spasm defects of vision of the nature of extreme astigmatism have been discovered. It seems, therefore, not unlikely that eyestrain may be the origin of these conditions. Little is known of the causation. The spasm usually persists until the child gets to be a year old, when it is outgrown. I have rarely seen it last through the third year. As the disease is not known in adult life it is evident that all patients recover. There is no known form of treatment, but in these children the general health should be carefully attended to. Proper nutrition should be secured; good air and good food, frequent bathing, and tonics, especially cod-liver oil and arsenic, are distinctly beneficial.

THE TWELFTH NERVE: THE HYPOGLOSSAL NERVE.

Anatomy. The hypoglossal nerve arises from a long group of cells which lies on the floor of the fourth ventricle in its lowermost part, where its walls approach and where it closes into the central canal of the spinal cord. (Fig. 198.) The nerve cells are large and multipolar. They send their axones ventrally through the medulla, passing at the side of the interolivary tract and pyramid and issuing between the pyramid and the olivary body on the ventral surface of the medulla; thence they make their exit from the skull through the anterior condyloid foramen of the atlas and pass to the muscles of the tongue. (See Fig. 177, *5*, page 440, and Fig. 199, page 476.)

Paralysis of the Hypoglossal Nerve. The hypoglossal nerve is rarely involved in lesions upon the base of the brain, though occasionally tuberculous or syphilitic masses have implicated it. In such cases the vagus nerve, which lies quite near it, is also involved. It is not affected in multiple neuritis.

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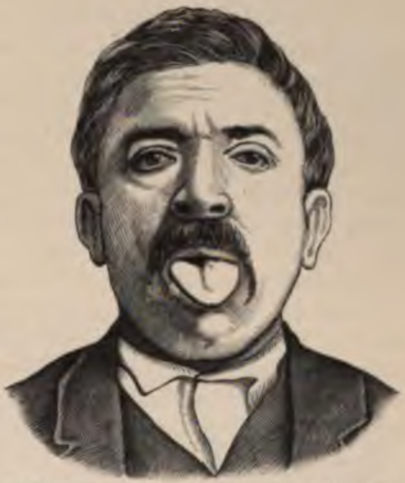
[1] Journal of Nervous and Mental Disease, 1897, p. 602.

A few cases of injury of the hypoglossal nerve have been recorded where this nerve has been divided by stab wounds or by gunshot wounds in the neck. Occasional attempts to cut the throat result in a division of the hypoglossal nerve. Cameron[1] described such a case with great care. The autopsy showed that the right hypoglossal nerve had been divided just internal to the bifurcation of the carotid artery. The cut ends were retracted, the central end was bulbous, and the distal end was found in a state of advanced degeneration. In this patient the right side of the tongue was paralyzed, had a flabby appearance, and presented three transverse wrinkles due to the atrophy; but the tongue could be moved in all directions, though imperfectly, and with difficulty toward the left. It protruded to the right side of the mouth in being put out, but there was no paralysis of taste.

FIG. 268.



Right hemiatrophy of the tongue. (Dejerine.)

The roots of the hypoglossal nerve are occasionally affected in disease located within the medulla. They pass close to the pyramidal tract and to the interolivary tract. These convey, respectively, motor and sensory impulses to and from the opposite side of the body; hence a form of alternate paralysis or alternate anæsthesia may be caused by a lesion which destroys one of these tracts and the hypoglossal root. Such lesions may be of vascular origin or may be new-growths.

The symptoms of paralysis of the twelfth nerve are inability to move the muscles of the tongue or to protrude this organ from the mouth. The result is that the tongue lies motionless within the mouth, its muscular fibres very often are in a state of fibrillary tremor and the tongue itself becomes atrophied, and the mucous membrane is thrown into folds with deep grooves between them which run lengthwise. (Fig. 268.) Inasmuch as the muscles of the tongue are closely interlaced upon the two sides, a unilateral hypoglossus paralysis does not entirely paralyze this organ. It can be moved in the mouth, and though the pronunciation of some of the lingual letters is interfered with, yet unless the paralysis is bilateral this is not very noticeable. In the act of protruding the tongue the healthy muscles elevate its posterior portion, pushing it outward more fully on the non-paralyzed side; hence the tongue deviates toward the side of the paralysis. Chewing and swallowing are not interfered with by unilateral hypoglossus paralysis, but are seriously impaired if the paralysis is bilateral. It is difficult to examine the tongue electrically on account of its great sensitiveness, but it may be possible to demonstrate a reaction of degeneration in its muscles.

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[1] British Medical Journal, 1901.

Paralysis of the twelfth nerve from disease of its nucleus may be produced as a part of the lesion of bulbar paralysis.[1] (See p. 624.)

The cortical centres for the movements of the tongue lie in the lower third of the motor area, and are joined to the nucleus by fibres which pass in the motor tract anterior in the capsule and median in the crus to the tracts for the arm and leg. These fibres decussate in the raphé of the medulla.

Partial paralysis of the tongue with deviation toward the paralyzed side is usually an accompaniment of hemiplegia, but in this case there is no fibrillary twitching and no atrophy in the tongue.

A spasmodic contraction of the muscles of the tongue (aphthongia) has been described by Charcot[2] and must have been known in the middle ages, as the best examples are found copied in stone in some of the gargoyles of the Notre Dame in Paris and other cathedrals in Europe. In this affection the muscles of the tongue are contracted, and as a result the tongue is forcibly thrust out of the mouth, either straight out if the spasm is bilateral (the common form), or toward one side if it is unilateral. The tongue may also be forcibly rolled in the mouth or thrust against the teeth. The spasm occurs, as a rule, in attacks, lasting a short time, but extremely annoying to the patient, and is not usually associated with any disease of the tongue, such as ulceration, smoker's tongue, or cancer. It has been regarded as a purely functional, possibly hysterical affection, but like all spasms may be reflex from some peripheral or central irritation.

The prognosis is good. The treatment should consist of nerve sedatives, especially bromides, chloral, valerian, and asafœtida.

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[1] Deut. Zeitsch. f. Nervenheilk, Bd. xiii., 4.

[2] Icon. de la Salpêtrière, vol. i. p. 87

CHAPTER XXXV.

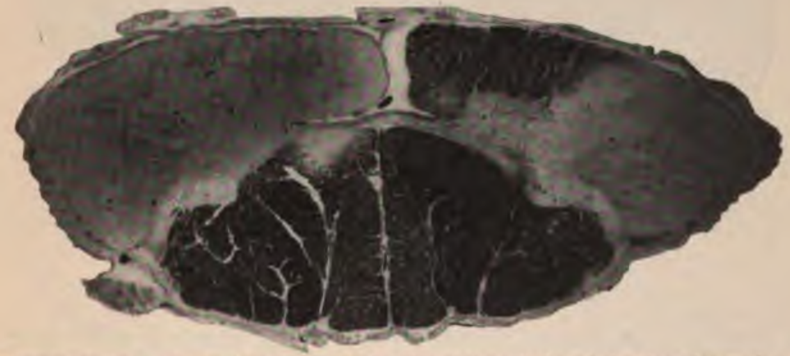
GENERAL DISEASES OF THE NERVOUS SYSTEM.

MULTIPLE SCLEROSIS.

MULTIPLE, or insular, or disseminated sclerosis is a disease which affects both brain and spinal cord together in varying degree, and is characterized by the production of small plaques or islets of sclerotic tissue scattered irregularly through the central nervous system.

Pathology. The sclerotic patches vary from a millimetre to several centimetres in diameter and are of irregular shape. There may be a hundred or more such plaques. They are rarely located in the cortex of the brain, but are found in the white matter beneath it, in the centrum ovale and in the corpus callosum, and in the gray matter of the basal ganglia; also in the pons, medulla, and spinal cord, and with less frequency in the cerebellum both in its white and gray matter. Sometimes the nerves, both cranial and spinal, appear to be invaded for a short distance from their origin.

FIG. 269.

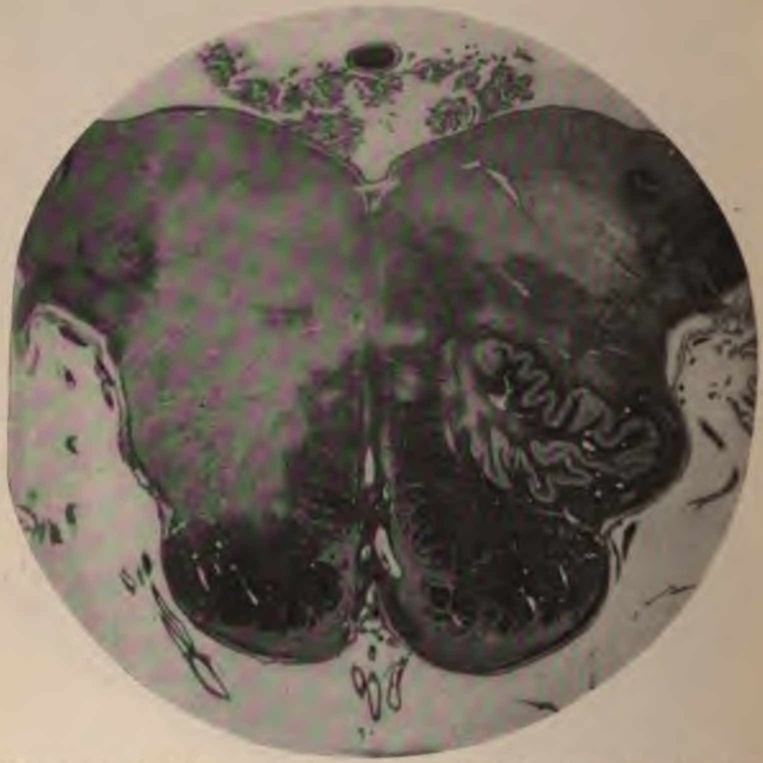


Multiple sclerosis. Section from the cervical region of the spinal cord, showing a large sclerotic patch on each side. (Spiller.)

The sclerosis differs from that occurring in other forms of sclerosis, as it is not limited to definite tracts. It does not lead to secondary degeneration in the various tracts in which it may happen to lie. This is because it spares the axones, though involving the medullary sheath of the neurones. In a fresh state the patches are pinkish-gray and more translucent than normal brain tissue. Some have a gelatinous appearance. They replace the brain tissue without increasing its volume, hence are not like small tumors. On section each plaque is sharply defined from the normal tissue about it, though the transition from abnormal to normal fibres is less sudden when a single fibre is followed along its course. It may be soft or hard and may in old cases cut like cartilage. There are no long columns of sclerosis, as in tabes or lateral sclerosis, but each islet is limited in extent.

On microscopic examination the sclerotic patch is seen to consist of very fine neuroglia fibres and glia cells, forming a feltwork of interlacing fibrils with dense infiltration with nuclei, and there may also be an increase of connective tissue. The fibres seem finer than ordinary neuroglia fibres, and are more abundant in the older cases, the early stage being characterized by the presence of small cells. In the older cases large spider cells are often found. There is usually some thickening of the bloodvessels. The neurones themselves are singularly free from degeneration, even in the midst of sclerotic patches; but occasionally cell bodies are found in various stages of degeneration when the plaque invades the gray matter, and fatty

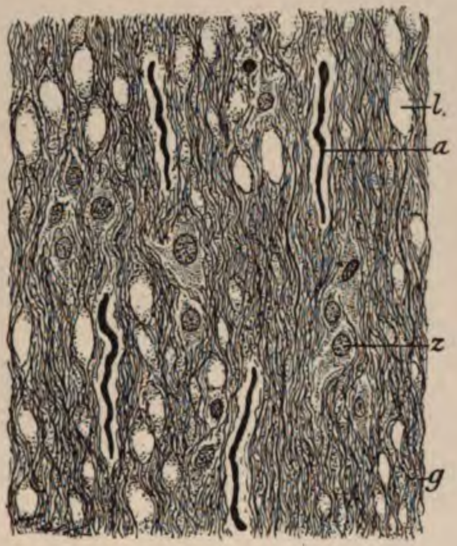
FIG. 270.



Multiple sclerosis. A sclerotic area implicates a large portion of the medulla oblongata. (Spiller.)

globules and granular corpuscles, the result of this degeneration, are then scattered through the plaque. Even by Nissl's method of staining there are found few or no evidences of degeneration in the neurones. In some regions a simple atrophy of cells has been seen. The nerve fibres passing through the islet of sclerosis appear to be squeezed, their calibre being reduced but their continuity preserved. The medullary sheath is reduced materially, and here and there an axone is broken and degenerated. The result of the degeneration of the myelin is seen in the presence of fatty globules and waxy granules in the nerve patches. But, as a rule, the axones are preserved and lie naked, adjacent to one another, through the plaque, and appear as if the medullary sheath had been dissolved off and absorbed. This peculiar appearance, first noticed by Charcot, has been confirmed by all subsequent observers and is characteristic of insular sclerosis. The preservation of the axone explains the absence of secondary degenerations in the various tracts. In a few recently reported cases, probably of very long duration, reliable observers have reported secondary degenerations in both sensory and motor tracts, but these are certainly the exception.

Fig. 271.

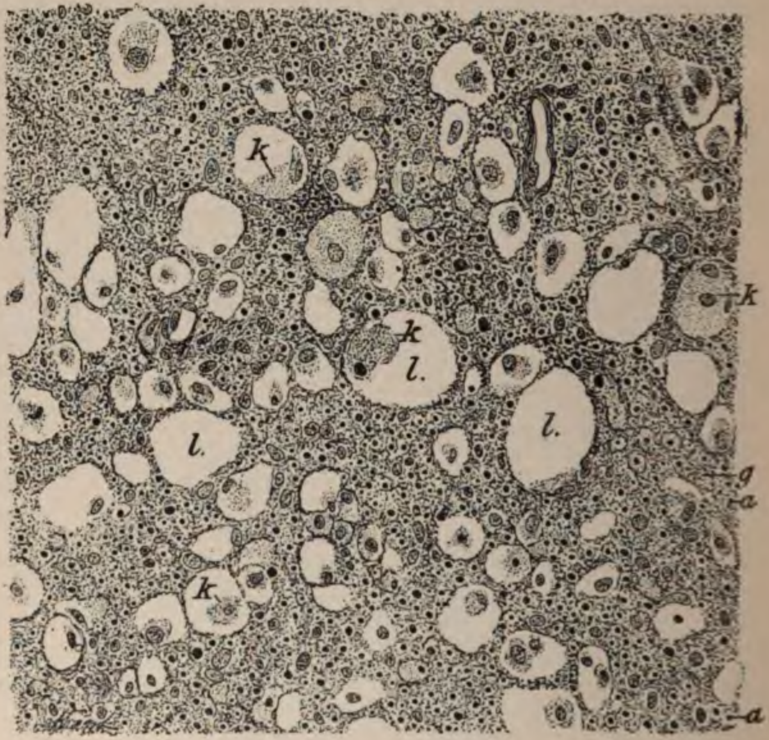


Multiple sclerosis. Longitudinal section through a plaque in the spinal cord. *g.* Thickened neuroglia. *l.* Empty spaces in the plaque. *z.* Large glia cells. *a.* Axis cylinder. (Schmaus-Sacki.)

The essential feature of the disease is therefore the production of a peculiar neuroglia structure which is associated with a disappearance of the myelin sheaths of nerve fibres. There are many theories in regard to this process, no one of which is generally accepted.

Strümpell believes that it is a congenital affection of the nature of gliosis, not unlike syringomyelia (*q. v.*), and that the nerve fibres are affected secondarily. He urges as proof the fact that it occurs in childhood. Schmaus suggests that instead of a process of degeneration of the myelin sheath there has been a defective development of myelin originally, and that the naked fibres have never developed a myelin covering, a possibility which is rendered more likely because of the fact that in the development of the nerve fibres the myelin appears later than the axone. Under these conditions it would be expected that the symptoms would appear in infancy instead of developing after the age of ten years or later.

Fig. 272.



Multiple sclerosis—areolar type. *g.* Glia. *l.* Spaces left by the absorption of nerve fibres. *k.* Granule cells in these spaces. *a.* Axis cylinder. (Schmaus-Sacki.)

Rindfleisch and others, Ribbert, Fürstner, Marie, Bartsch, and Schmaus believe that the process is vascular in origin, a toxic agent in the blood affecting first the vessels, then the medullary sheath of the nerves, and then leading to a secondary sclerosis. They affirm that the plaques lie about the vessels, which others deny. Others affirm that a lymph stasis in the lymph spaces about the bloodvessels is the primary cause; that these spaces are crowded with epithelioid cells which distend them, hinder the flow of lymph, and thus lead to swelling and degeneration of the glia cells and nerve elements, and finally to a sclerosis. But this theory has met with little favor, as the majority of observers regard this lymph stasis as a result and not as a cause, and when, under other conditions, such lymph stasis occurs it leads to a degeneration of the axis cylinder and not to one of the myelin sheath only. Charcot, Erb, Gowers, Leyden, Wernicke, and Thoma[1] hold that the disease is an interstitial or glia inflammation and that the myelin sheaths are destroyed secondarily. Leyden,[2] after a critical review, concludes that multiple sclerosis is the termination of a chronic interstitial inflammation. Redlich holds that it is a parenchymatous disease in the myelin, of toxic origin, and that the glia formation is secondary. André Thomas[3] affirms that it begins in the axis cylinder and affects some but not all of the fibrils of which it is made up. Its toxic origin is admitted by all, but the exact manner in which the toxic agent acts and the reason for the limitation of the lesion and the escape of the distal part of the axones is as yet a matter of mystery.

Etiology. Multiple sclerosis is a disease of youth, the majority of cases developing between the tenth and twenty-fifth year. No age, however, is exempt. Unger found nineteen cases in infants. The disease is rarely inherited, but two cases being on record in which a woman with the disease had a child who showed symptoms. It has no relation to syphilis. Cold and trauma were formerly supposed to be the active causes, and many cases have been recorded which render this probable, recent cases of Gaupp[4] and Blencke[5] being undoubtedly traumatic. Of late, owing to the investigations of Marie, more importance has been assigned to infectious and toxic agents, and now it is generally believed that the disease is usually a sequel of some acute infectious disease, typhoid, variola, malaria, scarlet fever, measles, whooping-cough, influenza, pneumonia, acute rheumatism, dysentery, cholera, or diphtheria. It has been known to develop in women after childbirth. I have seen such a case, and Balint[6] has reported a case in which after each of four successive confinements a rapid increase in the symptoms of the disease followed. Oppenheim has seen cases due to poisoning by metallic poisons, especially lead, zinc, tin, and mercury, and Etienne[7] has also reported one by carbonic oxide gas. Strümpell, who believes the disease to be of congenital origin and due to maldevelopment, ascribes little importance to these causes and holds that they are merely exciting causes when the defective nervous system is present.

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[1] Zur Path. hist. der Mult. Sclerose, Deut. Zeitschr. f. Nervenheilk., 1900, Bd. xvii. p. 265.

[2] Nothnagel's Spec. Path. u. Therap., Bd. x. p. 459.

[3] Rev. Neurologique, 1900, vol. viii. p. 490.

[4] Zur Aetiologie der Mult. Sclerose, Centralbl. f. Nervenheilk., June, 1900.

[5] Monatsschr. f. Unfallsheilk., Dec., 1900.

[6] Deut. Zeitschr. f. Nervenheilk., Bd. xvi. p. 437.

[7] Rev. Neurologique, 1900, vol. viii. p. 825.

Hoffman was not able to find any cause in more than one-half of 100 cases studied.[1]

Symptoms. It has been already stated that the lesions of multiple sclerosis affect the brain, brain axis, and spinal cord in different degrees in various cases, and therefore it is not surprising that three types of the disease have been described by writers according as symptoms referable to one of these parts predominate; but since in all cases which continue for a long time the terminal result is about the same, it is only necessary to mention these types as variations in the mode of onset. It must also be stated at the outset that great irregularity in the development and course of cases has been observed, and therefore Buzzard is quite right in holding that no typical picture of multiple sclerosis can be drawn. In fact, in many cases it is only by a process of exclusion of other forms of cerebral and spinal disease, and of hysteria in particular, that the diagnosis can be reached. It is also necessary to premise any statement of symptoms by calling attention to a fact admitted by all authors that all cases are characterized by occasional complete intermissions in the symptoms, one or many disappearing entirely for months, but returning later.

The ordinary type of case, first described by Charcot, is of very gradual development. The patient has some numbness and weakness in the legs, which slowly increases until he has a spastic or ataxic or even cerebellar gait, and stiffness and paralysis in the legs, with increased knee-jerks and ankle clonus and Babinski's symptom. There is usually a slight difficulty in the control of the sphincters which appears early. The skin reflexes are often lost. The gait is usually like that of lateral sclerosis, the feet being drawn along the floor and overlapping and the legs stiff and adducted. It may be a staggering gait like that of a drunken man, with irregular, uncertain steps. Sometimes it is a combination of these two gaits. The entire body sways in walking, which is due to an irregular contraction of the muscles of the trunk, producing what Oppenheim has termed "vascillation." At the same time a tremor, or rather a jerky, irregular action of the hands, appears, which is increased by effort both to hold them still and to perform any fine motion. The rate of the tremor is from five to seven per second. This is termed by the Germans an "intentional tremor." It does not occur when the hands are at rest, but gets worse and worse as they are used, the motions being more and more extensive, until finally the patient makes irregular, jerky motions and cannot attain his end. When he ceases to try the tremor stops. Thus he spills all the water from a glass in attempting to drink, and is unable to adjust his clothing or to write. The entire arm and forearm share in this tremor. Occasionally this tremor is confined to one side. The arms may become weak and stiff, but this is rare.

At the same time a marked defect in speech develops, known as scanning speech. It is a slow, jerky utterance of words, with pauses between words or between syllables, and a forced effort to pronounce, which leads to unusual accentuation and possibly to a clipping of words, so that they are not fully uttered. There may be also a tremor of the face and of the tongue, and tremor of the vocal cords has been seen by the laryngoscope. Sometimes there is a tremor or queer, jerky movement of the head which can be produced if the patient looks up or turns his head. This may occur, like a senile nodding, when the patient is at rest, the mere act of supporting the head being enough of a voluntary act to start the tremor.

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[1] Deut. Zeitschr. f. Nervenheilk., 1902, Bd. xxi. p. 1.

Nystagmus on lateral or upward movement of the eyeballs appears early, and later may occur when no motion is made. This is found in 75 per cent. of the cases. The pupils are often unequal and may be contracted, but always respond to light. Strabismus, due to a weakness of one or more ocular muscles, usually the externi, is an occasional symptom. This may only become evident on conjugate movements of the eyes to one side. Temporary attacks of blindness may occur, which are often followed by a permanent condition due to optic atrophy; in fact, optic atrophy may be, as in tabes, the earliest symptom, as shown by cases of Bruns and Stolling,[1] and is found in a large number of cases (52 per cent., Utthoff). It is usually a partial atrophy and causes a narrowing of the visual field or scotoma rather than total blindness. The outer or the inner halves of the disk are pale, or the entire disk may be white and sharply defined. There is also a change in color vision, red and green being lost before blue and yellow, which is the reverse of the order observed in hysteria. It is a peculiarity of the defects of vision in multiple sclerosis that they vary from day to day, at times disappearing entirely.

Vertigo is a common symptom, deafness is rare. Mental disturbance is frequently observed. There is a sense of well-being and a boastfulness without delusions which suggests paresis. There is a lack of control of the emotions. At times there is involuntary laughing, which is causeless and which annoys the patient, but cannot be controlled. There is imperfect memory and a manifest weakening in the power of reasoning and of judgment. In some cases (25 per cent.) attacks of epilepsy or of apoplexy with subsequent aphasia or hemiplegia occur. Paræsthesia is occasionally complained of, but anæsthesia is rarely observed. Pains sometimes give the patients distress.

In some cases the spastic paraplegia with atrophy of the muscles, loss of control of the sphincters, ataxia, trophic disturbances in the joints and skin, and bed-sores make it evident that the spinal cord is the part chiefly involved, and in these cases the other symptoms of bulbar and cerebral nature may be slow in developing, or may not occur before the patient dies.

In some cases the cerebral symptoms are the first to appear, especially scanning speech and hemiplegic attacks of temporary duration. It is in these patients that the diagnosis may be made of paresis or of cerebral softening, and only after months or years, when the bulbar and spinal symptoms develop, is the right diagnosis reached. The fact that other symptoms of paresis do not appear within a year of the onset and that the physical signs of paresis are absent may aid in the differentiation between the two diseases.

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[1] Monatsschr. f. Psych. u. Neurologie, 1900, Bd. vii, p. 89.

In a third type of cases the bulbar symptoms precede all others, and difficulty of swallowing, paralysis of the vocal cords, atrophy of the tongue, polyuria, and glycosuria, and attacks of suffocation may occur.

The course of the disease varies greatly. In some cases there is a slow but continuous progress in the symptoms, and after some years the patient becomes helpless, and finally dies of some intercurrent disease. In these cases the typical symptoms are so evident that the diagnosis is simple. In other cases there is a slow onset of the spinal symptoms which increase in intensity, and there are sudden attacks of an epileptic or apoplectic type, or sudden attacks of blindness which may subside. In still other cases there are remissions in all the symptoms which may be so complete as to lead to the hope of recovery, and recurrence takes place only after some months or even years. In other cases the onset is rapid, many symptoms appearing in the course of a few weeks, subsiding and recurring in a series of attacks. And lastly, there are cases which are said to go on to complete recovery by gradual subsidence of all the symptoms. Patients have died within two years of the onset. Others have lived for twenty years or more. It is thus evident that the multiplicity of the symptoms and their very irregular course makes any attempt to draw a picture of multiple sclerosis quite hazardous. It is also evident that a diagnosis can only be reached in the obscure cases by careful and long observation and by an attempt to eliminate all other organic and also some functional diseases.

The interest which unusual cases of disease excites has led to the report of many cases of multiple sclerosis in literature. As a fact, it is a very rare affection. In my clinic but 27 cases have been seen among 10,056 patients, and in my private practice the ratio is about the same.[1] Hence too great importance should not be attached to the space occupied by the disease in literature.

In one patient, a male aged thirty-five years, no cause could be ascertained. The first symptom noticed was a staggering gait quite like that of a drunken man, which was not increased by closing the eyes and was not accompanied by any change in the knee-jerks. After this had been present for some months the man's friends noticed a change in his speech which soon became typically scanning in character. Then it was noticed that there was a change in his manner, so that he gave the impression of being weak-minded and silly, laughed at everything which was said to him, and did not appear to appreciate his condition. Examination showed lateral nystagmus but no optic atrophy or ocular palsy and no actual paralysis. These symptoms had been coming on for a year at the time I saw him, and had not been intermittent, and were not affected by treatment.

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[1] See The Frequency of Multiple Sclerosis. Journ. of Nerv. and Ment. Dis., 1902, p. 288.

In a second patient, male, aged twenty-four years, no cause could be found. The first symptom was vertigo, which was increased by walking, and caused an irregular ataxic gait. Soon after a tremor began in the left hand which soon extended to the left leg and increased his ataxic gait, so that by the end of six months he staggered constantly and could hardly dress himself. This tremor was increased by any mental effort, by emotion, or by any active movement. Ten months after the onset he noticed a disturbance of vision which was not due to optic atrophy, but was produced by a lateral nystagmus, which was constant in looking at any object or on turning his eyes. About the same time typical scanning speech was noticed by his wife. This patient had no sensory disturbance, no paralysis, and no mental symptoms, but all his symptoms were rapidly increasing in intensity.

A third patient, a woman aged twenty-five years, had suffered for three years when first seen. Her symptoms began with tremor, first noticed in her head and neck, then in her hands, and finally in her body and legs, which was followed by the slow development of a spastic gait attended by increased knee-jerks. During the third year lateral nystagmus and scanning speech appeared. Her pupils and optic nerves were normal, and she had no mental symptoms, and her condition had remained stationary for some months when last seen.

A fourth patient, a woman aged thirty years, had suffered for four years. She had always been delicate, though she came of strong stock and had no illness prior to this one. Her symptoms began with stiffness and weakness of the legs, which in six months had developed into a spastic paraplegia with extreme contractures and loss of control of the sphincters. Then severe vomiting and indigestion began, which weakened her greatly, and for some time she had to be nourished by enemata. Then for three months she lay in a state of semiconsciousness, being fed by rectum, her bowels and bladder being evacuated artificially. Of this period she had no recollection, and it was with difficulty that she was kept alive. This condition and the paralysis were supposed to be hysterical, and an attempt was made by orthopedic apparatus to extend the contractured legs and to get her to walk. During this treatment, which lasted six months, her hands began to tremble, her speech became scanning, her eyesight became poor, and she saw double. Attempts to stand caused great shaking of the entire body and head, but became more and more successful, so that she was able to walk with the support of two persons, and recovered control of her sphincters. After three months of a fair degree of comfort her memory began to fail and her control over her emotions was much weakened. Then the intentional tremor increased rapidly, so that no voluntary movement was possible. There was no paralysis of the limbs when I saw her. There was a loss of knee-jerks, nystagmus, paralysis of the left external rectus, no loss of pupil reflex, vision was perfect, there was marked tremor of the intention type, and typical scanning speech. Her manner was childish and hysterical.

Diagnosis. The diagnosis from locomotor ataxia, spastic paraplegia, ataxic paraplegia, and amyotrophic lateral sclerosis is to be made (1) by the appearance of bulbar symptoms and cerebral symptoms, which do not occur in these diseases—*e. g.*, scanning speech, nystagmus, and lack of mental activity, and an emotional state; (2) by the lack of many symptoms which are characteristic of these diseases; (3) by the course of the case with remissions which do not occur in these affections. The diagnosis from cerebral disease, from paresis, multiple softening due to atheroma and brain tumor is to be made by the presence of intentional tremor and a scanning speech which does not resemble the trembling, indistinct pronunciation of the paretic; the absence of severe dementia; the absence of delusions; the absence of headache, vomiting, and choked disks; the preservation of pupil action; the intermittence of the symptoms, and the irregular course of the case. Toxic tremors and paralysis agitans never resemble intentional tremor and lack all other symptoms of multiple sclerosis. The diagnosis from hysteria may be at all times difficult, especially as hysterical manifestations frequently occur in the course of multiple sclerosis. Both diseases occur in young persons, though hysteria is more common in women and multiple sclerosis in men. Both diseases may follow an emotional shock, or a trauma, or an infectious disease. Both present a multitude of incongruous symptoms which come and go suddenly without apparent reason, and manifest great changes in character and course. Scanning speech and intentional tremor have been seen in hysteria. Nystagmus is never an hysterical symptom; optic atrophy is never hysterical; the peculiar oscillation of the head and body is not seen in hysteria, and loss of control of the sphincters is never hysterical. In the majority of cases a study of the individual, her heredity, nature, character, and her surroundings, and the discovery of the stigmata of hysteria will enable a diagnosis to be reached, though Buzzard, Oppenheim, and every specialist has been obliged to confess to occasional mistakes in diagnosis which were apparently inevitable. The knowledge of the possibility of such mistakes should intensify the effort to obtain objective proof of the existence of multiple sclerosis. So long as subjective symptoms only are present or symptoms which can be voluntarily simulated no one can be certain of a diagnosis.

There is a condition described by Westphal as "pseudo-sclerosis" which presents symptoms almost identical with multiple sclerosis, yet in which no lesions have been found. In this condition the mental symptoms, delirium and an apathetic state going on to moderate dementia, appear early and are more marked than in multiple sclerosis; the tremor is a slow one, the movements being but two to three to the second, and may occur during rest; all movements, even speech, are unusually deliberate; nystagmus and optic atrophy do not develop. Marie considers this disease of Westphal as a form of hysteria, but Strümpell, who has studied two cases, supports Westphal's contention that it is a neurosis, not hysterical in origin. It is not improbable that new histological methods may reveal a lesion as yet not discovered and as widespread as is multiple sclerosis.

The diagnosis of multiple sclerosis from Marie's form of hereditary cerebellar ataxia requires consideration, since both diseases develop in youth, both are attended by ataxia, nystagmus and tremor of the head, body, and limbs, and by mental failure. In multiple sclerosis some spastic paraplegia usually precedes the ataxia; this is not often of cerebellar type, and is attended by exaggerated reflexes. In multiple sclerosis optic atrophy is common, but it has not been observed in Marie's disease. The course of the disease is steadily progressive in Marie's disease, while in multiple sclerosis intermissions and remissions are the rule.

Prognosis. The prognosis in multiple sclerosis is unfavorable for recovery, but the condition does not make rapid progress, and as remissions in the symptoms occur spontaneously some relief from time to time may be promised. In a few cases the symptoms have subsided and have not returned for several years. The cases which have been reported as cured belong to this form. The earlier its development in life the better is the chance of its arrest. Even blindness has been known to subside, and, as the optic atrophy is never total, some degree of vision may be assured. The appearance of bulbar symptoms should always give rise to apprehension, as some cases of rapidly fatal termination from paralysis of deglutition or from respiratory failure have been recorded.

Treatment. It is essential that patients should avoid physical exertion, and a rest cure often helps greatly to arrest the disease. Even voluntary movements of the hands and too much talking may be avoided. The nutrition must be kept at the best possible point, fresh air, good food, tepid or alcohol sponge baths, massage, and all kinds of tonics being employed to increase the store of energy. As a rule, very hot baths are to be avoided, also cold shocks. A daily bath at 90° F., followed by rubbing, and salt baths at 95° F. are of much service. These patients do well at Nauheim and in water-cure establishments. General galvanization has been thought to be of use. In regard to the use of drugs, I have seen apparent benefit follow the use of arsenic, quinine, and salicin, and would urge these remedies, which may be used in succession two weeks at a time. Cod-liver oil is also of service. There is little effect to be expected from the use of mercury or of iodide of potassium. Nitrate of silver, once highly praised, has been abandoned.

CHAPTER XXXVI.

SYPHILIS OF THE NERVOUS SYSTEM.

SYPHILITIC affections of the nervous system may be produced by:

1. Syphilitic endarteritis.
2. Direct action of the syphilitic poison or its toxin upon the nervous system.
3. Syphilitic exudations in the meninges.
4. Syphilitic deposits in the brain, spinal cord, or nerves.
5. Hereditary syphilis.

These lesions are usually the result of a chronic syphilitic condition rather than of the primary effect of the poison. Hence, as a rule, they develop in the tertiary stage of syphilis, though occasionally they appear early.

1. Syphilitic endarteritis produces a progressive diminution in the calibre of the bloodvessels, both in the arteries, as Heubner has shown, and in the veins, as Rieder has shown. This causes a state of anæmia and malnutrition of the part of the nervous system supplied by the vessels affected, and leads to thrombosis followed by localized softening in the area cut off from its blood supply. The symptoms of this condition are, in the early stage, those of malnutrition of the brain or spinal cord and imperfect action in the regions affected. The patients present at first symptoms of neurasthenia and later slight temporary suspension of functions, such as temporary aphasia, numbness in one limb or in one-half of the body, a condition of weakness not amounting to paralysis in one limb or one-half of the body, double vision, or vertigo, or symptoms of transverse myelitis, followed by the secondary degenerations which it sets up. Stimulants to the heart relieve these symptoms, but after a time, if a thrombosis occurs, the symptoms of cerebral apoplexy or myelitis suddenly appear. After such an apoplectic attack a partial recovery may ensue, but as a spot of softening in the brain or cord is usually left, a complete recovery is not to be expected; hence the fact that a syphilitic lesion which can be removed by treatment is the cause of the apoplectic attack does not warrant a more favorable prognosis in such cases. It is not necessary to recount the symptoms of thrombosis in the brain or spinal cord, as they do not differ in syphilitic cases from those in other types. Hence the reader is referred to the chapters on vascular diseases of the brain and on myelomalacia. (See Figs. 211 and 212.)

2. The toxic effects of syphilis on the nervous system are manifested in some cases by general disturbances of function, which produce the symptoms of neurasthenia of various types. As a rule, in

syphilitic neurasthenia the symptoms appear to be worse toward evening, and insomnia is more persistent than any other symptom; hence in general neurasthenia with insomnia any syphilitic element must be looked for and treated before the neurasthenia can be cured. Neurasthenia may occur in syphilitic persons without any direct connection with the syphilis, which at the time may be latent. A test of treatment is the only one enabling a differential diagnosis to be made.

The most serious effects of the syphilitic toxins upon the nervous system are seen in the progressive degenerations which develop slowly many years after the initial lesion. These degenerations may appear in the cortex of the brain, leading to paresis (see page 556), or in the lateral tracts of the cord, leading to syphilitic spastic paraplegia (see page 278), or in the posterior sensory neurones in the spinal ganglia, leading to locomotor ataxia (see page 284). They may also appear as diffuse degenerative processes affecting both motor and sensory neurones, as in transverse dorsal myelitis (see page 347), in bulbar paralysis (see page 622), or in dementia. In all these diseases, while the causative factor may be evidently syphilitic, the symptoms produced, the course of the case, and the effects of treatment may differ in no respect from those in the ordinary non-syphilitic type. It is to be noted that in these degenerative affections antisyphilitic treatment has no power to alter the course of the case, and the prognosis in all is extremely unfavorable.

3. Syphilitic exudations into the meninges of the brain and spinal cord and syphilitic meningitis are exceedingly common as a sequel of syphilitic disease. From two to ten years after the infection gummy exudations may occur in any part of the brain, but are more frequent upon the base of the brain and about the crus and pons. The glue-like substance is deposited rapidly and extensively through the meninges, producing pressure upon the subjacent brain or upon the cranial nerves, or upon the spinal cord, and thus causing suspension of function in the parts compressed. Whether the syphilitic exudation takes the form of a soft or of a hard tumor, it produces the regular symptoms of tumor of the brain (see page 597) or of the spinal cord (see page 395). Gummy exudations may also occur in the nerves, the optic and oculomotor being the nerves most liable to this affection. (See Fig. 237; see also page 730.)

The pressure upon the vessels produced by syphilitic exudations adds to the complexity of the symptoms. The course of the case is more rapid in its onset than that of tumor of the brain or spinal cord, but resembles it in all other respects. From a study of the general and local symptoms it is not possible to determine that the tumor is syphilitic in origin. But in any case of tumor in the brain or spinal cord it is well to give the patient the benefit of the doubt, and if the symptoms subside rapidly under inunctions of mercury and large doses of iodide of potash the conclusion that the tumor was a gumma is justifiable. The prognosis is good in the majority of cases, though relapses are frequent.

4. Syphilitic deposits in the brain itself may be of the nature of small disseminated spots, producing chronic indurative or sclerotic processes or small regions of softening. The symptoms of this affection are identical with those of general paresis, and can only be distinguished from paresis by the result of treatment. (See page 562.) When these disseminated spots are located in the cerebral axis or in the spinal cord they produce symptoms which are identical with those of multiple sclerosis. (See page 700.) When they occur on or in the nerve roots or nerve trunks they cause symptoms of neuritis. In all cases antisyphilitic treatment meets with success.

5. Hereditary syphilis manifests itself in any or all of the lesions already described, and, as a rule, a combination of disease of the blood-vessels and of the meninges is found. It occasionally produces the symptoms of cerebral atrophy or the symptoms of multiple cerebrospinal sclerosis in children, and in either of these conditions this fact must be kept in mind, and if other signs of hereditary syphilis are discovered this element must be considered in the treatment. Many anomalies of development of the brain and some cases of hydrocephalus are clearly traceable to syphilis. The amaurotic family idiocy of Sachs is due to inherited syphilis.

It is evident then, from this summary that syphilis of the nervous system manifests itself by many different kinds of lesion and produces many different types of symptoms. Much attention has been given to the subject during the past few years, the works of Fournier,[1] Rumpf,[2] Oppenheim,[3] and Nonne[4] having forced its importance upon the notice of physicians. It must, however, be admitted that there is nothing pathognomonic about any disease of the nervous system to convince one that it is syphilitic, and hence it seems sufficient in this chapter to refer to the various chapters in which the different diseases traceable to syphilis are described. The treatment of syphilis of the nervous system is described on page 325.

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[1] La syphilis du cerveau. Paris, 1879.

[2] Die syphilitische Erkrankungen der Nervensystems. Wiesbaden, 1887.

[3] Oppenheim. Lehrbuch der Nervenkrankheiten, 1902.

[4] Syphilis und Nervensystem, Berlin, 1902.

CHAPTER XXXVII.

PACHYMENINGITIS AND MENINGEAL HEMORRHAGE.

PACHYMENINGITIS or inflammation of the dura mater, may be limited to the external or to the internal surface of the dura, or may involve both surfaces at once.

PACHYMENINGITIS EXTERNA.

Pachymeningitis externa occurs as a result of fractures of the skull, or follows any disease of the bones of the cranium, such as caries, especially after ear disease, syphilitic exostoses, tumors invading the bones, and erysipelas of the face.

In septic cases it may lead to the formation of an abscess between the dura and the bone. The only symptoms produced are those of the original causative disease, and local pain, which may be increased by pressure and by percussion. In these cases trephining will lead to the evacuation of the pus.

It may take the form of a connective-tissue proliferation, causing a thickening which is permanent.

In a few cases a firm adhesion between the dura and the cranial bones, with the production of bony deposits and a solidification of the diploë, has been found. This may occur in early life and lead to the development of chronic headache with many neurasthenic and hysterical symptoms which resist all forms of treatment.

PACHYMENINGITIS INTERNA.

Etiology. This disease may result from the same causes as pachymeningitis externa, especially from trauma, or may be a sequel of it. It is never possible in life to distinguish sharply between the two conditions. It may be a sequel of sunstroke. It has been found in many cases of chronic insanity, especially in paretic and senile dementia. It has been ascribed to chronic alcoholism. It has been found in cases of purpura and in persons dying of various infectious and wasting diseases; a causal connection, however, has not been established. It may be due to syphilis.

Pathology. The result of an inflammation of the inner layer of the dura is the production of a thin vascular membrane covering it like a veil. The vessels in this are extremely delicate and rupture easily. As a result a hæmatoma forms, flat and thin, but of sufficient size to compress the brain beneath and to cause symptoms. Some pathologists believe that in cases of hæmatoma the hemorrhage from congested vessels occurs first, then the clot becomes organized and adherent to the dura, and then new vessels form in it. Bevan Lewis believes this to be the invariable case in the chronic insane. The new membrane may be limited in extent or quite large. It may lie on any part of the dura. If the condition goes on for some time several layers of new tissue may form until the dura is several times its ordinary thickness. In such a thickened series of layers flat hæmatomata are often found. The age of the various layers can be determined by their degree of hardness and vascularity. In the older, deeper layers there may be thin deposits of bone. In the superficial layers there is occasionally an adhesion to the pia. This is the condition found in chronic alcoholism and in terminal dementia.

Symptoms. The disease may be present for years as a chronic condition, causing dull headaches only or slight difficulty in thinking, with impaired memory, and may not be diagnosticated. It is by the occurrence of the hemorrhages which produce symptoms of a temporary apoplexy that the suspicion may be awakened of a pachymeningitis hemorrhagica. Thus an alcoholic subject who had had a sunstroke and had at various times suffered from blows on the head complained for some weeks of severe headaches, and suddenly had an attack of left hemiplegia attended by vomiting and a short general convulsion. In the course of two weeks all signs of the hemiplegia subsided and it was evident that no destruction of brain tissue had occurred. His headaches continued and four months after the first attack a second with right hemiplegia and aphasia occurred which proved fatal. The autopsy showed an extensive pachymeningitis interna with a large recent hæmatoma on the left side and evidences of an old one on the right side. In any case where apoplectic symptoms occur and subside rapidly the possibility of hæmatoma must be considered. During the attack and immediately after it the diagnosis of apoplexy is the only one possible. The pupil is dilated on the side of the lesion, the head and eyes may turn toward the lesion, and a rigid state of the limbs may exist for some hours after the attack.

The course of a case in pachymeningitis is sometimes very slow. The patient may suffer for several years from occasional attacks of severe headache lasting several days and then gradually subsiding, but leaving him with disagreeable cerebral sensations and a sense of inability to do his work. He may be aware of difficulty in fixing his attention upon business or matters of importance, and may suffer from difficulty in recollecting recent events. He may manifest all the symptoms of cerebral neurasthenia with occasional attacks of vertigo and prostration. As time goes on he may have sudden attacks of unconsciousness, with or without twitching of a general character in the limbs, or with a true Jacksonian epileptic attack, and after such an attack he may be much prostrated for ten days or two weeks, being confused mentally, or almost stuporous, or being very irritable and restless. Then all these symptoms may subside and he may return to a state of apparent health and even be able to go back to business. But even when apparently well he will be known by those who are most intimately associated with him to show some changes in disposition and character which are unnatural. After a period varying from six months to a year the headaches may reappear, convulsions may be repeated, and inability to think and remember may again become noticeable. Attacks of hemiplegia, or of hemianopsia, or of aphasia may follow, and the patient may again be reduced to a state of invalidism. It is seldom that two remissions occur, but they have been observed. Finally, all the symptoms become intensified, the headache becomes continuous, the mental dulness becomes more marked, and the patient is obliged to go to bed, where he lies in a state of semi-stupor for weeks or even months. In this condition he can be aroused, will answer simple questions, but shows no interest in his surroundings or in his family, and is indifferent to his own condition. He will take food when it is offered, but seems to live in a dazed condition, and is very likely to neglect ordinary cleanliness. He sleeps heavily in the daytime and is wakeful at night, and very often has a muttering delirium during the night; at times starting up in a state of alarm; at times crying out as if in pain, and at times being restless and desirous of getting up and going out. A general condition of motor weakness gradually develops, even when the patient does not show the traces of apoplectic attacks. His movements become uncertain and ataxic, with considerable tremor of the extremities and even of the entire body. In getting up out of bed he will do so in a feeble manner, with much trembling and shaking of the trunk, and it seems difficult for him to retain his balance or to stand alone, oscillating movements of the body and of the head being constant. When he is up he seems unable to decide what to do, whether to remain standing or to sit down, and acts in a dazed manner. There is rarely any loss of sensibility to touch, temperature, or pain, but sometimes the patient appears to be deaf, and in some cases the eyesight is affected.

The patient may remain in this condition for several weeks, gradually losing strength and weight, and finally dying of some complication, such as obstinate constipation, cystitis, or bed-sores. A progressive emaciation and a condition of extreme anæmia or of nephritis are not uncommonly observed in the course of the disease.

Optic neuritis has been occasionally found. In these cases brain tumor may be suspected and cannot be excluded.

Treatment. It is so seldom that one can make a diagnosis of pachymeningitis interna hemorrhagica that it is almost futile to speak of treatment. Ice to the head, or, better, applications of the actual cautery, may relieve the headache. Purgatives of a drastic kind may also be used. When an attack of an apoplectic nature occurs it is to be treated as a cerebral hemorrhage. It is rarely possible to operate for the removal of such a hemorrhage at the time of its occurrence. There may be indications which justify an operation later, as in the following case:

A child, aged nine years, had a severe fall on the head, that was followed by coma lasting several hours, after which a left hemiplegia was discovered. This subsided entirely in the course of two weeks, but he continued to have severe headaches, and after two months attacks of Jacksonian epilepsy on the left side, beginning in the hand, commenced. These kept up with increasing frequency for six months, not being controlled by bromides. He was then operated upon by McCosh, a large bony flap being made and free access to the brain being had. The inner surface of the dura over the motor area of the brain was covered by a thin, velvet-like layer of tissue which bled freely on being touched. This was adherent to the pia over the middle third of the posterior central convolution, but was easily stripped off. It was removed by a curette, the hemorrhage, which was trifling, being controlled by pressure, and the dura was united and the wound closed. The child recovered from the operation and had had no return of the Jacksonian fits at the end of two years.

The operation was undertaken on the belief that a cortical traumatic hemorrhage had left a cyst which was causing the epilepsy and which might be removed. The discovery of the condition of pachymeningitis interna hemorrhagica was a surprise. The ease with which the hæmatoma was stripped off led to the idea that it was merely the remains of a flat clot, but microscopic examination showed it to be an organized membrane with capillaries identical with that found in pachymeningitis interna.

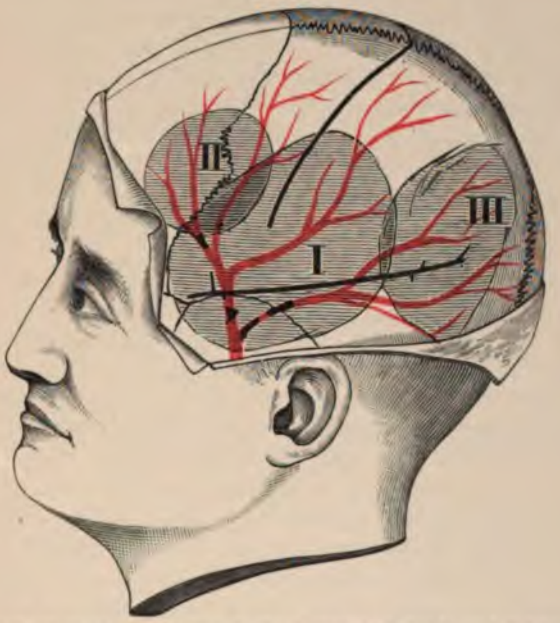
MENINGEAL HEMORRHAGE.

Hemorrhage from the middle meningeal artery occurs either with or without fracture of the skull as the result of trauma. In Fig. 273 the sites of the most common varieties of meningeal hemorrhage are shown. These hemorrhages are between the bone and the dura. The symptoms of such hemorrhage are general and local. After a blow or fall on the head, with or without apparent fracture, for fracture of the internal table may not be evident, the patient becomes comatose and is found to be paralyzed on one side, with the pupil on the side of the injury very widely dilated and not responsive to light. The coma may not be deep, the patient being rather in a deep stupor, and this may continue with varying degree until the pressure is relieved by trephining. Or consciousness may be regained slowly, and then either (1) aphasia, or (2) hemiplegia, or both together, or (3) hemianæsthesia, or (4) hemianopsia are discovered according to the position of the hemorrhage, which causes a diffuse compression of one hemisphere. Such cases should be trephined as soon as the diagnosis is made and the torn vessel tied and the clot removed.

Hemorrhage inside of the dura, either from its vessels or from some vessel in the pia mater is also caused by trauma with or without fracture of the skull. Hemorrhages which occur in infants during labor, from delayed or instrumental delivery, are usually inside the dura and result in a large clot spread out on the surface of the brain. In some cases the vessel torn is a large one and the clot is rapidly formed, produces great pressure, and death follows.

In other cases the vessel is a small one and the clot extends slowly; the symptoms then gradually increase during several hours or even days until the bleeding ceases, and then subside more slowly still and only in part. In infants the existence of such a pressure by a large clot may prevent the process of development of the brain and lead to atrophy and sclerosis. (See Chapter XXVII.) The general symptoms of pressure are diminishing consciousness to the degree of stupor, or even coma, headache, a slow pulse, vomiting, and possibly difficulty of respiration, and a rise of temperature. The local symptoms are those of compression of some part of the hemisphere. This is usually on the side of the injury, but occasionally on the opposite side. A dilated pupil on the side of the lesion, deviation of head and eyes to that side, and aphasia, symptoms of paralysis, or loss of sensation or of sight on the opposite side, are the local symptoms, and their

FIG. 273.



The meningeal artery and its relation to the fissures of Rolando and Sylvius. I. Area of a hemorrhage from the main artery, causing hemiplegia and aphasia. II. Area of a hemorrhage from the anterior branch, causing mental dulness and aphasia. III. Area of a hemorrhage from the posterior branches, causing sensory aphasia and hemianopsia.

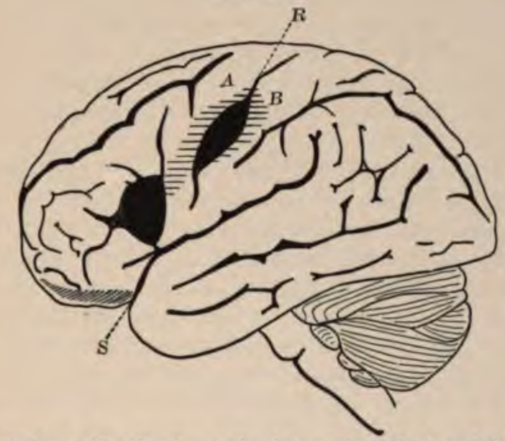
value is to be estimated as in cases of cerebral hemorrhage. When these symptoms presist for more than two weeks there is evidence of continued pressure, and it is not likely that a clot large enough to cause them will be absorbed, hence it must be removed. The treatment, therefore, should be by trephining. The following case is a good illustration of this condition:

A physician, aged forty years, was thrown from his carriage on August 17, 1889. He was slightly stunned by the fall, but had no wound, and was able to help his wife, who appeared to be injured, to reach home. For several hours he seemed to be suffering merely from bruises, and was able during the afternoon to converse with a patient and to administer a hypodermic injection. In the course of the evening, however, he became delirious, then stupid, and for the following three days he lay in a semi-comatose condition. On the morning after the injury he was found to be completely hemiplegic on the right side, and aphasic. When, after a week, his consciousness had fully returned, it appeared that the aphasia was purely motor, as he could understand what was said to him, and could read. The hemiplegia was attended by a partial anæsthesia, the paralyzed limbs felt numb, were less sensitive to touch and to pain, but acutely sensitive to cold and to heat. In this condition he remained until December, when he was brought to New York and admitted to Roosevelt Hospital.

On examination, the patient, a large man, was unable to say anything, even "yes" or "no," the vowel sounds being the only sounds produced by effort. He evidently understood questions, and attempted to reply by gestures and by trying to write with his left hand. It was noticed, however, that he was mentally dull, so that the result of tests of muscular sense were not satisfactory, though he could be tested for tactile and pain senses, which were found to be impaired on the paralyzed side. He was emotionally unstable, laughing too readily, and at times appearing to be much depressed. Ophthalmoscopic appearances were normal. Sight and hearing were normal. The right hemiplegia was partial. He could turn his eyes in all directions, but could not turn his head to the right. His left pupil was one-third larger than the right pupil, but both reacted normally. His face was slightly flat but not paralyzed, and his tongue protruded straight. His arm was almost totally paralyzed, the only motion possible being a slight abduction at the shoulder. His leg could be moved a little at the hip and knee, and when held up by two persons he could drag the leg forward a little, but could not stand alone. The hand was flexed and pronated, the leg was extended; both were very rigid, and all the deep reflexes were greatly exaggerated, so that wrist and finger clonus, as well as patella and ankle clonus, were easily produced. He controlled his sphincters perfectly. There were no scars upon the head.

It seemed probable that, as a result of the fall, there had been a rupture of a small vessel from which very slow hemorrhage had taken place; the vessel was thought to be a vein rather than an artery, because of the very slow development of the symptoms. The situation of the clot was thought to be upon the surface and not within the left hemisphere, and it was located upon the posterior part of the third frontal convolution and over the anterior central convolution in its middle third, as shown in the diagram. A flat clot in such a situation might fail to compress the face area and could produce an absolutely total motor aphasia, while a subcortical or capsular clot could hardly produce total permanent motor aphasia and paralysis of the arm without producing paralysis of the face and tongue. On the strength of this diagnosis it was thought best to operate.

FIG. 274.



Meningeal hemorrhage. The situation of the clots causing aphasia and right hemiplegia. Removal resulted in recovery. (Starr and McBurney.)

On December 13, 1889, McBurney trephined the skull. The trephine was applied at a point one and seven-eighths inches behind, and seven-eighths of an inch above the external angular process of the frontal bone, and the opening was then enlarged by the rongeur forceps upward and backward, the dura being laid bare over an ovale area three by two inches. The dura did not pulsate. On opening the dura the pia was found to be very œdematous and discolored, and the surface of the brain was separated from the dura by a space half an inch in depth, and did not pulsate. The clot was seen lying beneath the pia upon the posterior part of the third frontal convolution and extending over the anterior central convolution in a thin layer (marked in lines on the diagram) into the fissure of Rolando, which was filled with a larger clot lying in the situation shown in the diagram and extending downward so as to fill up and distend greatly the cul-de-sac at the lower end of the fissure. The clot had not covered the lower third of the anterior central convolution and had not reached the upper quarter of the fissure of Rolando. The brain, at a distance of an inch about it, appeared to be healthy and pulsated, but the parts of the cortex on which the clot lay were pulseless and stained a yellowish-red. After the pia had been incised the clot was removed little by little by fine sponges, at least a drachm of partly organized clot being taken out of the fissure of Rolando. The retraction of the brain from the skull was even more evident when the operation was complete. The operation was done under strict aseptic precautions, and was not followed by any rise of temperature. The wound was dressed in the open method, being packed with gauze and drainage tubes being inserted. On renewing the dressings three days subsequently to the operation it was found that the entire surface of the brain was pulsating normally and that the brain surface presented a normal color and appearance. After a week the drainage tubes were removed, and after three weeks the wound had entirely healed, the level of the scalp at the bottom of the cavity being an inch below the normal level.

On the evening of the day of operation the patient said "yes" and "no" for the first time since the injury, and after that time his recovery of speech and of power was progressive. After a few days it was noticed that he was much more intelligent and no longer emotional. Power in his leg began to increase very soon, and two months after the operation he was able to walk with a cane. The return of speech was slow but continuous, and it appeared as if he were learning a new language. He repeated words after another until he had learned them. He talked in monosyllables for the first three months, then he began to put two words together, and then used short sentences of three or four words. Fifteen months after the operation he was able to practise medicine in his native town, was able to walk without a cane, could name at once any object shown to him, but in talking used only a few words at a time, not speaking fluently, wrote with his left hand, and had it not been for the loss of power in the right hand, might have been considered fairly well. The paralysis of the right hand remained, and with it a marked diminution of tactile and pain senses, two points being felt as one when two centimetres apart on the tips of the fingers. The muscular and temperature senses were perfect. He could move the arm and forearm in all directions with much force, and could supinate the hand, but movements below the wrist were very slight, the fingers being flexed and rigid. The reflexes were much less exaggerated than before the operation, clonus not being elicited excepting at the ankle: the head could be turned in any direction, and the pupils were equal. In this condition he remained for ten years, until he died of pneumonia.

CHAPTER XXXVIII.

CEREBRAL MENINGITIS.

Epidemic Cerebro-spinal Meningitis. Endemic Meningitis; Hydrocephalus. Septic Meningitis. Secondary Meningitis. Tuberculous Meningitis. Syphilitic Meningitis.

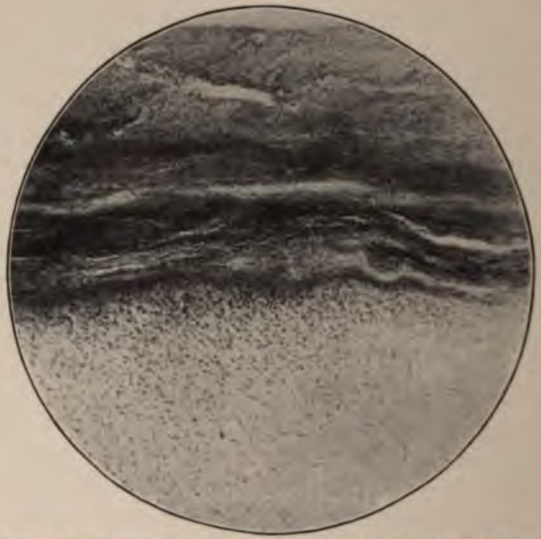
MENINGITIS is an inflammation of the pia mater. It is an acute disease in the majority of cases, and is then of bacterial origin. There are several classes of cases:

1. Epidemic cerebro-spinal meningitis, or spotted fever, which is due to the invasion of the pia by the diplococcus intracellularis.
2. Endemic meningitis of infants due to infection by various micro-organisms.
3. Septic meningitis, which occurs with wounds, fractures, and operations, or after otitis media, and is due to the invasion of the pia by streptococcus or staphylococcus; and secondary meningitis which occurs as a complication of pneumonia, ulcerative endocarditis, empyema, typhoid and typhus fever, influenza, the eruptive fevers of childhood, erysipelas, and in fact any form of disease due to a micro-organism. In these cases the germ of the disease attacks the pia mater, and has been found in it. The list of bacteria which have been detected in the pia is increasing daily, a large variety having thus far been found. There is no special bacillus of meningitis, though various investigators have isolated various forms and made a claim to its exclusive causative action. Thus Still has found a diplococcus allied to that of cerebro-spinal meningitis, Weichselbaum has described a meningococcus intracellularis, and Babes a micrococcus lanceolatus.
4. Tuberculous meningitis, due to the direct invasion of the pia by the tubercle bacillus.
5. Syphilitic meningitis, due to an exudation of gummy material into the pia mater.

The entire pia of the convexity and of the base may be involved in the inflammation, but certain forms of meningitis appear to select certain localities. Thus in the epidemic form the entire pia, both of the brain and of the spinal cord, is invaded. In the septic and secondary forms the pia of the convexity is particularly affected, and that of the base may escape. In these cases the process may be localized in a small area. This occurs especially after wounds, after otitis media, and nasal disease. In meningitis of infants the base is particularly affected and hydrocephalus is a frequent result. In tubercular meningitis the pia on the base alone may be the portion inflamed, hence this form has been named basilar meningitis. In syphilitic meningitis the pia on the base near the posterior perforated space and about the crus cerebri or at the sides of the medulla and pons is usually affected. It is evident that this difference in location will cause a great difference in the symptoms of the various types.

Pathology. After a short period of hyperæmia the congested pia becomes covered with an exudation of serum, lymph, fibrin, and pus. The serum infiltrates its meshes and collects between the pia and the brain, making the latter œdematous. It also collects below the arachnoid, lifting this so that it appears to contain cysts. The fibrin is deposited in flakes on and in the pia, rendering it less transparent than normal and covering it and the brain with a thick opaque lymph. The pus is infiltrated through the meshes of the pia, and as it increases in amount fills up the space between the convolutions along the lines where the pia dips down, then it collects in the sulci, and finally may form masses of a yellowish-green color, covering the surface and filling up its irregular spaces. The cortex of the brain on which this serous and purulent exudation lies soon becomes affected; in fact, in every case one has to deal rather with a meningo-encephalitis than with a simple meningitis. The cortex is greatly congested. Wherever the pial vessels dip into the cortex, serum, lymph, and pus are exuded, and small hemorrhages and little collections of pus are found everywhere in the cortical layers. Occasionally large areas of softening or little abscesses are found in the brain. The pia covering the cerebellum and the cranial nerves is similarly affected, and in cases where

FIG. 275.



Acute meningitis of the convexity of the brain. The thickening of the pia, its infiltration with the products of inflammation, and the invasion of the cortex by cells is seen.

the process extends downward the pia along the entire length of the spinal cord may be inflamed and covered with lymph and pus.

The pia extends into the vetricles, and hence a similar process goes on there, resulting in an effusion and consequent distention of the ventricles with serum, which soon contains flocculi of fibrin and pus. The cerebro-spinal fluid contains similar flocculi, cells, and free bacteria, and hence by lumbar puncture and examination of the fluid obtained the diagnosis of meningitis can always be made.

In cases in infants which last for more than a week a distention of the skull commences, due to this accumulation of fluid in the ventricles, and this results in a separation of the sutures, which goes on until a hydrocephalic shape of the head and a great increase in its size is produced.

The collection of fluid in the lateral ventricles only occurs when an adhesion has formed in the membranes, closing the foramina and obstructing the free communication between the ventricles and the subarachnoid space. The choroid plexuses of the ventricles and the epithelium covering them secrete the cerebro-spinal fluid, which is not a mere transudation from the blood. Being constantly secreted it must be drained away and absorbed. This drainage occurs through the cerebro-spinal foramen and the two lateral foramina of the fourth ventricle which establish a communication between the ventricles and the general subarachnoid space. The lateral ventricles communicate by the foramen of Monroe with the third ventricle, and this empties into the fourth ventricle by way of the aqueduct of Sylvius, the minute size of which secures a slow drainage. If any obstruction occurs to such a flow, either by compression of the aqueduct or by closure of these foramina, an accumulation of fluid will occur. In meningitis, particularly of the base, there occurs an adhesion of the membranes or an effusion of lymph which closes these foramina. Drainage being impossible fluid accumulates in the ventricles and causes hydrocephalus. Barlow[1] believes that in many cases of congenital hydrocephalus and of chronic hydrocephalus developing slowly without symptoms of meningitis, a slight attack or a low grade of meningitis has occurred at the outset which has not been noticed or has recovered. In the majority of cases of meningitis in infants, if death does not take place before the third week, hydrocephalus appears. (See page 536.)

In tuberculous meningitis there is rarely an exudation of pus. There is a deposit of miliary tubercles through the pia, both diffuse and in conglomerate masses. These are small white egg-shaped bodies, usually first appearing along the bloodvessels and always found on their walls. An exudation of serum, lymph, and fibrin occurs, and these form a thick mucilaginous material in which the miliary tubercles are imbedded. This process is always much more marked about the base of the brain, where the many irregular spaces are filled with the new material. It may extend to the convexity, however, and then the exudation is more evident along the sulci and fissures, especially in the fissure of Sylvius. The miliary tubercles may be found scattered widely over the entire extent of the pia and in the ventricles. The ventricles are always distended with cloudy serum in which fibrin and blood are found. This distention may be extreme and may produce hydrocephalus. The exudation surrounds the nerves upon the base and causes a neuritis. A diffuse encephalitis of the cortex or a hemorrhagic encephalitis, either localized or general, may accompany tubercular meningitis. Masses of tuberculous material in a state of cheesy degeneration are also found in the membranes or in the brain in some cases. (Fig. 229, page 586.)

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[1] Allbutt's System of Medicine, vol. vii. p 493.

Syphilitic meningitis is characterized by a serous and fibrinous exudation in the membranes attended by a deposit of mucilaginous material of a gummy nature which is semi-translucent, thick, and viscid, and which surrounds the nerves and fills up the spaces on the base or in the sulci of the convexity. It begins to appear along the bloodvessels, but soon becomes extensive. It is not attended by the production of pus, and there is not often any exudation of serum into the ventricles. The exudation invades the cranial nerves, especially the optic and oculomotor, and causes a degenerative neuritis. The pia is thickened by a production of connective tissue which causes its firm adhesion to the dura and to the brain. Areas of softening on the cortex are commonly found. (See Fig. 237, page 593.) The characteristic syphilitic endarteritis is always present in the arteries of the base. (See Fig. 211, page 494.)

Lumbar puncture, first done by Quincke in 1891, is a valuable aid to diagnosis in all forms of meningitis. The patient is placed on the left side, the legs being drawn up and the body bent far forward. A line is drawn between the posterior-superior edges of the ilia, which will pass across the space between the third and fourth lumbar vertebræ. The lower edge of the spinous process of the third lumbar vertebra is found, and puncture is made by a long hypodermic needle just below this in children, and 1 cm. to one side of it in adults. The hypodermic needle is thrust in between the laminæ, and should be pointed a little upward and inward. It can be felt to penetrate the membrane, and then its point can be moved more freely than in the solid tissue. The syringe is then unscrewed from the needle and the fluid allowed to run out. It must never be exhausted by suction. A small trocar may be employed in place of a hypodermic needle. A few cubic centimetres only of the fluid is to be taken in a test tube. The pressure under which it flows is to be noticed, and if it spurts out of the needle the internal pressure is abnormally high. This occurs in meningitis. If it will not run the patient's head and body may be elevated, and thus a few drops may be obtained. It is usually easy to obtain 10 c.cm. Normal cerebro-spinal fluid is clear, straw-colored and contains a few cells and not more than 0.2 per cent. of albumin. In meningitis it is cloudy, contains a little blood, flocculi of fibrin, polynuclear leucocytes, pus, and micro-organisms. It usually contains more albumin than normal. On standing the cerebrospinal fluid coagulates, and the coagulum forms more quickly in cases of meningitis and is firmer than in health. It is the existence of pus, leucocytes, and bacteria which are the most important diagnostic signs of meningitis. Cultures should always be made of the fluid. The finding of streptococcus, staphylococcus, pneumococcus, or micrococcus intracellularis, or of tubercle bacilli, makes it certain that a meningitis is present. In tuberculous meningitis mononuclear lymphoid cells are also found, while in the other forms polynuclear cells are more common.

Symptoms. 1. **Epidemic Cerebro-spinal Meningitis.** The disease, though occasionally occurring in epidemic form, particularly in winter and spring, both in cities and in the country, is more commonly seen as a sporadic affection, and is quite rare. Children are more susceptible to the infection than adults. Unwholesome, dirty surroundings, malnutrition, and overexertion, either mental or physical, are supposed to make one more susceptible. Though infectious, the disease is not contagious, and is not carried in clothing.

The period of incubation is not determined.

The symptoms appear suddenly, with a chill, temperature of 104° F. or higher, full rapid pulse, headache, pains in the back, and vomiting, which is projectile. The patients feel very ill, are restless and irritable, and soon complain of light and sound. Stupor and coma come on rapidly. Within a day or two of the onset the headache becomes very intense, and stiffness in the back of the neck appears, which is very painful. The head is held rigid or is drawn back, the back also becomes stiff, so that children can be raised from the bed by lifting the back of the head, and a state of opisthotonus develops, attended by great pain in the back and limbs and extreme hyperæsthesia to all forms of sensation. The slightest touch causes great pain. In addition to the rigidity of the muscles there are occasional spasms of a clonic or tonic kind in the extremities, the face, too, being drawn, and tremor in the limbs may be observed. The pupils are dilated, and sometimes unequal, and do not react to light. Grinding of the teeth and strabismus are early symptoms.

Within two days of the onset delirium of an active character begins, and this continues, alternating with stupor and coma, for a long time. The mind is always clouded and filled with illusions, and the patients have no memory of what has occurred when they recover. The delirium may at times be wild, and is always worse at night and when the temperature rises. After the third day an eruption appears in the majority of cases, though it is not an invariable symptom. It is petechial, diffused over the entire body, but deeper, and purple in spots. Herpes appears on the lips early; erythema, pemphigus, and gangrene of the skin have all been seen in various cases in addition to the petechiæ. Any scratch on the skin leaves a dark red stripe, the *tâche cérébrale* of Trousseau. The gastro-intestinal functions are affected from the start, nausea, vomiting, diarrhœa, or more commonly, obstinate constipation being present. The urine contains albumin, casts, and sometimes blood. It is sometimes retained, but may be passed involuntarily, as are also the feces. The spleen is enlarged. Contractures soon develop, and the limbs are flexed until paralysis ensues and they relax. The reflexes are increased.

There are some cases of a malignant type which end within the first two or three days. Many cases prove fatal within a week. If the patients do not die of fever or of heart failure within a week of the onset they usually have a remission in the symptoms, but these return again, and the case may go on for many weeks with varying intensity. There is no typical temperature curve, and great variations are possible. In one patient who recovered I saw a rise on five occasions during two months to 106° F., the ordinary variation being between 101° and 103° F., with occasional falls to normal. A sudden rise or a sudden fall is unfavorable. The temperature may be intermittent. The pulse may be rapid, especially in children, or it may be as slow as forty per minute. It is frequently intermittent. The respiration is irregular, at times of the Cheyne-Stokes variety, and attended by sighing and yawning. It is sometimes difficult to feed and care for those patients, as they resist in their delirium all attempts at nursing, and rapid and extreme emaciation results. The most prominent symptoms during the long illness are headache, delirium, stupor, insomnia, rigidity of the muscles, especially of the neck and back, hypersensitiveness to touch, to sound, and to light, and a gradual loss of voluntary power.

The cranial nerves are almost always seriously affected. Strabismus, which develops early, persists. It is often attended by nystagmus. Vision may be obscured by keratitis, and optic neuritis and atrophy may develop and finally render the patient blind, though in two patients I have seen a partial recovery of sight, sector-like defects in the visual field remaining. Deafness is a common symptom, due to otitis media or to an affection of the auditory nerve, and may be permanent, leading to deaf-mutism in children. Difficulty in swallowing and in articulation are observed in many cases. General convulsions may occur from time to time in the course of the case.

The most permanent of all the symptoms is a state of mental apathy and indifference. Even after the delirium and stupor have passed off the patients may show no mental activity, may fail to control the sphincters, may not recognize their parents, may have no interest in persons or events, and may talk with great difficulty. I have seen these symptoms remain for several weeks after the temperature had become normal. In the patients who do not succumb to general exhaustion or die of a complicating pneumonia, arthritis, or cystitis, or bed-sores, there is a very slow return to health, and sometimes it is six months or a year before the mental and physical symptoms have entirely disappeared. The severity of the disease depends somewhat upon the character of the epidemic, some epidemics being very fatal, in some the mortality being 75 per cent., in others being as low as 20 per cent. In some epidemics the tendency to complications seems to be great.

In sporadic cases the prognosis depends on the intensity of the symptoms, but it is never well to give up hope, as very bad cases may recover.

The treatment consists in supporting the strength by fluid food and alcoholic stimulants, given often and freely; in the application of ice to the head and spine in bags, or of ice-water in coils; in cool bathing when the temperature exceds 102° F.; and in the free use of sedatives, of which bromides, phenacetin or acetanilid, sulphonal, and trional, and in many cases codeia or morphine, in small dose frequently repeated, are the best. Strychnine should never be given. Some authors recommend ergot in large doses. It is questionable whether mercury or iodide of potassium are useful, though some writers urge their use. In the stage of recovery iodide is useful in doses of twenty grains three times a day for adults.

2. **Meningitis in Infants; Hydrocephalus.** Infants appear to be much more liable to the infection of meningitis than adults, and in them the picture of the disease differs greatly from that already presented. Both sexes are equally liable. The majority of cases occur during the first and second years of life, though no age is exempt. Barlow's statistics point to a greater susceptibility between the third and sixth months. Catarrhal diseases of the intestines, bronchi, or nose precede the onset in many cases. A history of injury to the head is obtained in not a few.

The symptoms develop acutely, and are well marked within three days of the onset. Vomiting, convulsions, retraction of the head, screaming, and irritability of temper are the first symptoms, and are soon followed by sleepiness and languor, by bulging of the fontanelle, by strabismus and nystagmus, and by some inequality or abnormality in the action of the pupils, by champing movements of the lower jaw, lips, and tongue, and by extensor spasms of the limbs, and in some cases by opisthotonus. Paralysis of the face and limbs is occasionally seen, but is rare. The infant undoubtedly suffers from great pain, as is shown by the constant crying and screaming, which are kept up at night and by day, and by pulling its hair. This is an early symptom, and subsides later when the stage of hydrocephalus is reached. Irregularity of respiration is often noted, and the pulse is either very fast or very slow. The skin is flushed and shows the mark of any irritation as a broad red line or spot. The temperature varies greatly between 100° and 106° F. The abdomen is often retracted; constipation is more frequent than diarrhœa. One of the most noticeable symptoms is the rapid emaciation, and these children become mere skeletons in the course of a couple of weeks. Hydrocephalus usually develops in the second week of the disease. In addition to the increase in the size and change in the shape of the head, there is often a slight bulging of the eyeballs, and strabismus appears. This condition may persist for a long time, or it may gradually subside as the child recovers.

The course of a simple meningitis may be rapid, and a fatal termination may occur within a week. This rapid course occurs chiefly in the vertical and sometimes in basal cases. More often the symptoms persist a long time and the case runs a course which extends over several weeks. The variation is so great in different cases that no average duration can be stated. There are some cases which continue even for four or five months, and then recover. That a large percentage die must be admitted. Authorities make the mortality from 60 to 80 per cent. It has been thought by some that children never recover from meningitis, but this is not my experience. I have seen some well pronounced cases in the Baby's Hospital which terminated in recovery. Even cases which develop hydrocephalus, if this is moderate, may come to a standstill and gradually get well. These patients may be left blind or deaf, or they may show a spastic gait for years, or they may show mental defects, but nevertheless they recover from the acute meningitis. Occasionally during the stage of improvement, or even after recovery appears to be complete, sudden death occurs. There is no disease, therefore, in which the prognosis is more uncertain.

The treatment must be quite similar to that described in the epidemic form. Leeches behind the ears, ice to the head and neck, or a cold coil, antipyretics, and cool sponging are of service in keeping down the temperature. The child should be nourished as well as possible on milk properly modified to its age, and on broths. It may be necessary to feed by gavage if there is much vomiting. There are no drugs which act on the bacteria, and hence there is no means of decreasing the pathological process. In some cases iodide has been of service, and it may be given if the stomach will retain it in one to three-grain doses four or five times a day.

It is not to be forgotten that some cases are secondary to otitis media; hence if any evidence of this is present free drainage must be secured by incising the drum or by operating on the mastoid. Cases are now frequently reported where the mastoid operation, followed by trephining, has resulted in the evacuation of a small collection of pus and has been followed by recovery.

3. **Septic and Secondary Forms of Meningitis.** The symptoms of a septic meningitis always develop after some one of its causative conditions has been in progress for some time. Hence they are preceded by the symptoms of a cranial fracture or operation, an otitis media, a chronic nasal disease, or an abscess, or erysipelas of the face. If, in the course of any of these affections the patient has a sudden chill, with rise of temperature, rapid pulse, severe continuous general headache, and delirium or convulsions, and if thus an illness begins which is evidently septic, is attended by a stuporous mental state, somnolence, vertigo, vomiting, and a fever running from 101° to 104° F. daily, there is every reason to believe that a septic meningitis has

developed. The general symptoms mentioned usually appear before any local signs of brain disease. And these will depend largely on the area which is affected by the meningitis. Thus, in fractures of the skull which lie over the motor or sensory areas there appear local spasms, or aphasia, or paralysis, or disturbances of sensation, first hyperæsthetic conditions, and later loss of sensation. After otitis media there may be no local signs of meningitis, as the temporal lobe on the base has no known function; but if the disease advances upward on the left side a sensory aphasia may appear. If the meningitis is on the base the cranial nerves may become involved; nystagmus, strabismus, twitching, or paralysis of the face, grinding of the teeth, and optic neuritis develop. These sometimes occur after otitis media. If the meningitis starts from nasal disease there are no local signs excepting loss of smell and a greater degree of mental disturbance than in the other cases.

The same general symptoms developing in the course of any of the infectious diseases will indicate that meningitis has begun as a complication. The headache which is always present in meningitis is severe and causes great agony. The patient grasps the head and moans with pain even during his delirium and stupor. Children pull the hair, knock the head upon the pillow, and scream, even in their sleep. The sudden piercing cry of pain which wakens the child is known as the cry of meningitis. The pain varies in intensity, and is worse at night or when the temperature rises. The delirium is another characteristic symptom of meningitis. It is sometimes a low muttering delirium from which the patient can be momentarily aroused, but he does not answer questions intelligently. At times it is a more active delirium, the patient wishes to get out of bed, to dress and go out, or is wildly excited, and struggles with his nurses and has to be tied in bed. In the intervals of delirium there is a stupor which may deepen into coma. The fever of meningitis is high. It does not fall or rise suddenly, as in thrombosis of the lateral sinus, or in pyæmic states, but runs a course between 100° and 104° F., and occasionally the patient may have a chill. The pulse, which at first was rapid, varies as the disease goes on. It often becomes intermittent and irregular. It may for a few hours be slow, even 40. It does not vary with the temperature. The general hyperæsthetic state of all the senses is very characteristic of meningitis; the patient begs to be left alone in the dark, starts at slight noises, and cannot endure to be touched or examined. There are twitchings of the muscles, which are increased by tapping, and there is often an increase in all the tendon reflexes. The neck and back become stiff and painful early in the course of the disease. And various cranial nerve symptoms indicate that the effusion on the base has begun.

The pupils are usually contracted at first, do not dilate in the dark, and do not react to light. Later they may become dilated, are often unequal, and the condition of the pupil varies from day to day. In some cases the pupil contracts to light, but immediately dilates again, and continues to change in size; this is termed hippus. Ptosis, paralysis of some of the ocular muscles, with strabismus and nystagmus, soon develop. Optic neuritis occurs in about 60 per cent. of the cases. The facial nerves may be irritated, causing a twitching of the facial muscles and trismus or paralysis. As the case progresses general convulsions may occur, followed by coma. Monoplegia or hemiplegia may develop at any stage. There is an irritation or paralysis of the vasomotor system shown by blueness of the extremities, a reddening of the skin when scratched or rubbed, and profuse sweating. There is obstinate constipation. The urine may be retained or may dribble. It often contains albumin, and is of high specific gravity and scanty. The spleen is enlarged. In the later stages there may be a tendency to bed-sores.

The duration of a meningitis may be from four days to two weeks. In rapid cases all the symptoms appear in great intensity, the temperature rises high, the pulse is weak and rapid, coma comes on, and the patients have Cheyne-Stokes respiration, and die either in coma or in convulsions. In slow cases the stage of irritation gives place to a stage of paralysis and the patient is exhausted by the fever.

I have seen several cases which ran a slower course and in which symptoms persisted for four or even six weeks. In these cases remissions in the symptoms, with subsequent exacerbations, took place; but the general symptoms of fever, headache, somnolence, and progressive emaciation were constant. The formation of abscess in the brain or the occurrence of thrombosis of one of the sinuses, either of which may be a complication, makes the prognosis hopeless.

The diagnosis of meningitis is to be made by an examination of the cerebro-spinal fluid obtained by lumbar puncture (see page 720), and this should be tried in all doubtful cases.

The diagnosis from abscess of the brain and from thrombosis of the lateral sinuses has been considered on pages 576 and 582.

The prognosis in septic and secondary meningitis is always serious. Patients rarely recover. Nevertheless, the possibility of recovery has been proven in many cases in every variety of the disease, and hence it is always legitimate to hold out some hope. It is also to be remembered that there are many cases presenting serious nervous symptoms quite similar to those of meningitis in their character, but not quite equal to them in severity, which pass off in the course of four, six, or eight days. I have seen many such cases in connection with otitis media. I have seen some in children complicating other diseases or appearing alone. We do not know what the pathology of such cases is. They have been ascribed to cerebral hyperæmia, to serous meningitis, to œdema of the brain, to non-purulent encephalitis, and to direct toxæmia of the brain. The only means of diagnosis of these cases from meningitis is by watching their course and observing the recovery. Hence in the early stage of supposed meningitis of a mild type the prognosis must be guarded, as the diagnosis may be wrong.

The treatment of septic meningitis or of secondary meningitis should begin by an active interference with the cause. Fractures should be trephined and a possible source of infection sought and free drainage opened. In middle-ear disease an early exploration of the mastoid cells and prompt opening of the skull, with drainage, are often necessary. In abscess of the brain and sinus thrombosis operation affords relief. In other cases operations are impossible. Absolute quiet in bed, ice to the head, brisk purgatives, leeches to the mastoid region or temples, blisters to the neck, and the free use of narcotics, the coal-tar products, and morphine are to be employed. The temperature is to be controlled by cool sponging rather than by baths, as it is impossible to move the patient into a tub. Antipyrine and phenacetin may help to reduce it if it rises above 103° F. The heart may require stimulation, and for this caffeine is better than either alcohol or strychnine. The delirium may be somewhat diminished by the free use of bromide and chloral. If the persistent headache is soon followed by optic neuritis it is probable that a large effusion has occurred, and it is possible that lumbar puncture may reduce this. In some cases it has succeeded and an improvement has followed. From 50 to 100 c.c. of fluid have been withdrawn daily for several days under strict antiseptic precautions.

4. **Tuberculous Meningitis.** Children between the ages of two and fifteen years are more commonly affected by tuberculous meningitis than are adults, though no age is exempt.

The symptoms of this form of meningitis are numerous and differ so widely in different stages of the progress of the case that all authors prefer to group them in periods. These are (I) a period of incubation, when the symptoms are slight and rather indefinite, but really well marked when one looks backward and contrasts the condition with that of previous health; (II) the period of irritative symptoms; (III) the period of paralytic symptoms and coma.

I. In the period of incubation the child, without reason, ceases to gain weight and begins to lose, is fretful and uneasy, shows little desire to play, and cannot be amused or diverted for any length of time, very often manifesting a marked change in disposition and unusual likes and dislikes. At night it does not sleep soundly, wakes on slight noises and cannot be put to sleep, or groans and cries in sleep, and sometimes grinds the teeth. In the daytime it may be drowsy. Vomiting, occurring without apparent cause and recurring in attacks, is usually an early symptom, and is attended by considerable prostration. Headache soon appears, being complained of by children who can talk and being shown by crying, tearing the hair, and beating the head in infants. During this period the temperature is usually 100° or 101° F. at night, but nearly normal in the morning, the pulse is more rapid than usual, and the respiration regular, but attended by yawning and sighing. The appetite is poor, the bowels are often constipated; occasionally there is diarrhœa. The urine is usually increased in amount, and may show high specific gravity and albumin in small amount. The prodromal stage is longer in tuberculous meningitis than in other forms.

As tuberculous meningitis is secondary to tuberculosis elsewhere in about 80 per cent. of the cases, some symptoms referable to the lungs, or intestines, to the joints, or the bones, or the spine, to the brain, or to the lymphatic glands, are to be detected when the child is carefully examined. These symptoms should lead to the diagnosis, even in the stage of incubation. This stage may last from four days to three or four weeks.

II. The stage of irritative symptoms appears suddenly, usually with a general convulsion, followed by stupor. The child, who has gradually become weaker and sicker, complains of most intense headache, is hypersensitive all over, and cannot endure being touched, shuns the light and noise, and cries when disturbed. It is somnolent, but cries out in sleep, grinds its teeth, and rolls its head, wearing off the hair from the occipital region. Then, without warning, it has a general convulsion, which may be repeated at intervals. When it comes out of the convulsion it is stupid and cannot be aroused, but cries when disturbed. There is usually some rigidity of the neck, which increases rapidly and extends to the spine, so that the entire body is stiff, and after a time the head is drawn backward. Sometimes the limbs become rigid and contracted. The reflexes are exaggerated. The pupils are contracted and do not respond to light, and are often of unequal size. There may be nystagmus and hippus. The temperature begins at this time to rise at night to 102° F., the pulse becomes faster and is now occasionally intermittent and irregular, and the respiration is irregular, a series of inspirations, each deeper than the last, being followed by a sigh and cessation for a few seconds. This stage may last for two or three days, or even ten days, during which time the stupor deepens, and the child sleeps more and more continuously, and it is harder to arouse or interest it. Delirium is a common symptom, the child talks to itself, occasionally screams out, and may not know its parents. Toward the close of the stage muscular twitching is frequent and partial convulsions may occur; the abdomen is retracted, *tâche cérébrale* is well marked, and an ophthalmoscopic examination shows choked disks. The skin is dry, and ulcers may form about the ears or on the buttocks. In spite of the severity of the symptoms, however, the child may at times seem for an hour or more to be better, may be conscious and alert, and have no pain. But these intervals become infrequent as the last stage approaches. In this stage lumbar puncture usually shows the presence of tubercle bacilli in the cerebro-spinal fluid.

III. The stage of paralysis or coma comes slowly. The child gets worse, the stupor deepens, so that it is impossible to arouse it, and paralysis of some of the cranial nerves appears, causing strabismus, facial paralysis, or difficult swallowing, and general paralysis of the limbs, or hemiplegia. The pupils are now dilated and fail to act to light; choked disk appears, if it has not before; tubercles may be seen in the choroid; the opisthotonus is more marked, and the abdomen is retracted. The temperature begins to rise in the morning as well as at night, the pulse becomes very rapid and weak, respiration is irregular and labored and of the Cheyne-Stokes character. It is almost impossible to feed the child, and emaciation is rapid. The urine is retained or is passed involuntarily. The coma deepens, and the child dies of exhaustion or in a convulsion. The duration of this stage is from two days to a week, depending on the nutrition and inherent strength of the child. A quarter of the cases die in the first week after the second stage begins, a quarter in the second, a quarter in the third, and the remainder within eight weeks. In infants whose fontanelles are not completely closed a condition of hydrocephalus is liable to develop and to go on increasing until death. There is no doubt that almost all cases die, and we have no means of arresting the progress of the disease. Yet cases have been reported in which recovery has occurred, even when tubercle bacilli have been found in the cerebro-spinal fluid. Treatment is palliative only, the various symptoms being treated as they arise. It has been proposed to open the skull and drain the base. This has been done once with apparent success by Ord and Waterhouse, but their example has not been followed by others. As all cases are hopeless, such a measure, though theoretically of little value, may be worth a trial. It failed in one case under my care.

The following case is a good illustration of the course of the disease: Girl, aged two years, of healthy parents, began to have slight attacks of sudden loss of consciousness without convulsions, on November 7th, and complained of headache, nausea, and vomiting. During the first two weeks these symptoms continued, and she was very fretful, dull, and stupid, emaciated rapidly, and had a temperature of 100° F. at night. During the third week the temperature reached 102° F. at night and did not go below 100° F. in the morning; her pulse was 120, regular, respiration regular; she had much headache, retained little food, cried out in sleep, and was drowsy and dull. On November 28th she had a convulsion limited to the right side of the face and right arm lasting an hour and a half, after which she slept for two hours and had a temperature of 104° F. There was no paralysis after the convulsion, but she failed to recognize her mother and nurse and kept her hand on the left side of her head, with signs of pain. Her pupils were dilated, reacted sluggishly, and dilated in the light, being undulatory. Her optic disks were normal. During the following week she failed steadily, being in a state of stupor, and finally of coma. Her temperature varied from 101° to 104° F., pulse became 140 and intermittent, respiration sighing, irregular, and finally Cheyne-Stokes. She had one right-sided convulsion. Her pupils were contracted when she was let alone, but dilated widely when the eyes were open and undulated. The optic disks became cloudy and finally choked. The face was flushed, and there was *tâche cérébrale*. . The abdomen became retracted. There was no paralysis, but the knee-jerks, which in the first weeks were absent, became exaggerated, and ankle clonus was obtained. She died on December 5th. The autopsy showed tubercles scattered over the pia, especially on the base and in the fissures of Sylvius. There was much serum under the pia. The ventricles were greatly distended by cloudy serum. There was some œdema, but no inflammation of the brain and no apparent cause for the limitation of the convulsion to the right side, though there was more serum over the left hemisphere, about the motor zone, than elsewhere. In this case it was evident at the autopsy that no operation would have had any effect to relieve the condition.

5. **Syphilitic Meningitis.** The symptoms of this form of meningitis develop more slowly than those of the other forms, and are more easily and successfully treated. They resemble the symptoms of brain tumor, but are more rapid in their progress. The patients suffer from general sensations of discomfort in the head, from headache, which is often occipital, sometimes frontal, sometimes diffused over the entire head, and is usually worse toward evening, and keeps them awake in the night. They become nervous, cannot control the emotions, and cannot think quickly or keenly; hence they are unfitted for busidess. They are subject to attacks of vertigo, and may at times vomit unexpectedly without preceding nausea. After a time dimness of vision may be noticed, and an ophthalmoscopic examination shows an optic neuritis; or a third nerve palsy may suddenly occur, with ptosis, external strabismus, and immobility of the eyeball and pupil; or a facial palsy or sixth nerve affection, with internal strabismus may occur. It is more rare for the other cranial nerves to be affected, although intense neuralgia of the face, deafness, and difficulty of swallowing have been observed. After some one or more of the cranial nerves on one side have become paralyzed a hemiplegia of the opposite side not infrequently develops, not complete, but attended by increase of reflexes. Sensory disturbance is rare. Polyuria is a very common symptom in these cases. Occasionally a cerebellar gait has been noticed. In one of my cases general convulsions occurred several times before treatment began to produce an effect. The symptoms, therefore, are quite like those of tumor on the base of the brain. Sometimes the gummy exudation is in the Sylvian fissure and causes compression of the bloodvessels supplying the cortex. In these cases sudden attacks of aphasia or hemiplegia may occur.

The meninges of the convexity are occasionally involved in a syphilitic meningitis, and in these cases symptoms resembling a localized brain tumor, attacks of Jacksonian epilepsy, or of aphasia, followed by monoplegia, may occur. The headache may be localized at one spot and may be attended by tenderness on percussion of the head. Mental symptoms are prominent in these cases, resembling those of paresis, but with dementia more evident than delusions. The symptoms may be very irregular and intermittent, as in the basilar type, severe on one day, slight on the next; they may vary in their location, and they are rarely as permanent and continuous as in brain tumor. The occurrence of an optic neuritis is the exception in meningitis of the convexity. The difficulty of diagnosis between a specific meningitis of the convexity, paresis, and brain tumor is so great that in every case where the question of diagnosis arises it is well to try antisyphilitic treatment. If this fails operative treatment may be considered, for successful removal of gummy tumors has been recorded.

The various symptoms named develop gradually in the course of a month or six weeks, increase in intensity, and prostrate the patient completely. But when treatment is begun they soon begin to subside, and finally may entirely pass away, leaving the patient quite well.

In cases of long duration, where the nerves or the brain have been seriously compressed, and where degeneration in the nerve trunks or in the brain tracts has been produced, the recovery is much delayed or may be incomplete. Thus in one of my cases which showed paralysis of the sixth, seventh, and eighth nerves on the left side, and a marked cerebellar gait, with staggering toward the left, the cranial nerve palsy recovered, the gait became natural, but there has remained at the end of three years a sense of uncertainty of position and some vertigo on rapid walking and an exaggerated knee-jerk on the right side. In another case, after a period of occipital pain, worse at night, the right sixth and seventh, then the fifth, then the third nerves were paralyzed, and then a left hemiplegia appeared. These symptoms subsided under treatment, but after a few months deafness in the right ear and a marked staggering to the right side, with polyuria, developed. These also subsided under treatment, and for ten years this man has been well and able to work, but suffers from vertigo.

The prognosis is fairly good in syphilitic meningitis, as treatment promptly causes the absorption of the exudation; but it must not be forgotten that symptoms due to an actual destruction of cerebral tissue are permanent.

Treatment, no matter at what stage in the course of syphilis, should be by means of inunctions of mercury. One drachm of blue ointment is to be well rubbed into the body daily, and the absorption is increased by a daily bath in water of a temperature of 106° to 108° F. for half an hour. This bath may be followed by cool sponging to prevent too great sweating and taking cold. The application of mercury should be to a different part of the body each day, and this part should be bandaged, so that any ointment left on the skin may be absorbed and not rubbed off by the clothing. I prefer this method to that by hypodermic injection of corrosive sublimate, though this may be resorted to in some cases. Pills of mercuric iodide, red or green, usually derange the stomach and their effect is less rapid than that of the inunctions. After the mercury has been given for one week iodide of potassium is to be begun in addition to the mercury.

It is my rule to begin with a dose of twenty-five grains three times a day, as thus the coryza which is caused by small doses is avoided. The amount is then increased rapidly, one grain at each dose until three hundred grains daily are being taken. On reaching a dose of fifty grains it is better to increase the number of doses than to increase the size of each dose. If the iodide is given well diluted in water, in some gaseous mineral water or in milk, it can usually be taken for several months. The mercury should be continued with the iodide until marked improvement is manifest or until salivation is caused. This should, of course, be prevented by the use of a mouth wash of chlorate of potash, and by cleaning the teeth after each meal and after each dose of medicine. Usually three ounces of blue ointment are sufficient but more may be needed.

The general strength of the patient should always be kept up to the best point, every possible means being used to promote health. An out-of-door life in good country air, moderate exercise, the best food possible, and a generous diet, with moderate use of stimulants and tonics of all sorts are to be advised. The benefit of a course of treatment at hot springs or in a sanitarium is partly due to the more healthful surroundings than city life affords.

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